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Brit. J. Ophthal. (1958) **42**, 65.

COMMUNICATIONS

VASCULARIZATION OF THE PRIMARY OPTIC PATHWAYS*

BY

J. FRANÇOIS, A. NEETENS, AND J. M. COLLETTE

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FIRST we will study the macroscopic or extra-tissular, and afterwards the microscopic or intra-tissular vascularization of the primary optic pathways. The latter has been especially studied by microradiographic examination of sections 0·1–0·5 mm. thick after injecting Heyden's thorotrast.

(A) MACROSCOPIC (EXTRA-TISSULAR) VASCULARIZATION OF THE PRIMARY OPTIC PATHWAYS

The central retinal artery, though making use of the optic nerve to reach the retina, supplies only the latter.

The optic nerve itself is supplied on the one hand by the central artery of the optic nerve—an axial system derived from the ophthalmic artery—and on the other hand by peripheral vessels localized in the pia mater and dependent not only on the ophthalmic artery but also on the ciliary arteries and sometimes even on the extra-optical part of the central retinal artery (Fig. 1).

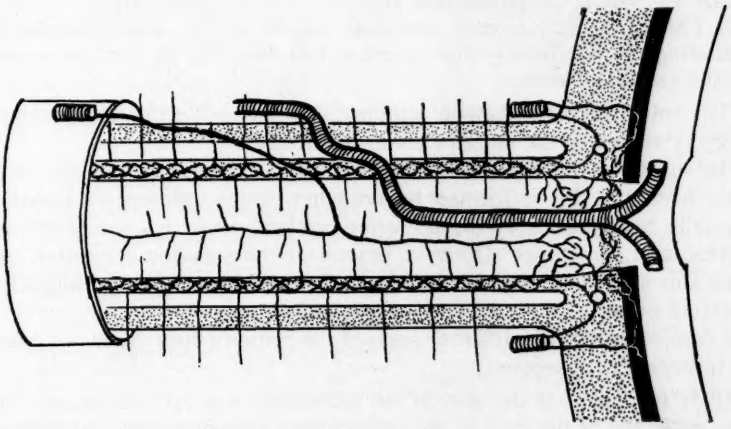


FIG. 1.—Intra-orbital part of the optic nerve (François and Neetens, 1954, Fig. 9, p. 486).

The central artery of the optic nerve, seen only at the level of the intra-

* Read at the combined meeting of the Society of British Neurological Surgeons and the 36th Annual Meeting of the Irish Ophthalmological Society in Dublin, May 16–18, 1957.

orbital part of the nerve, divides into two branches, anterior and posterior, both of which are localized in the nerve axis.

The peripheral system, the arteries of which reach the surface of the optic nerve perpendicularly and divide dichotomously, is reinforced in front, at the level of the lamina cribrosa of the sclera, by the vessels of the circle of Zinn-Haller derived from the short posterior ciliary arteries, and behind, at the level of the intracanalicular and intracranial sections of the optic nerve, by a marked increase in the number of large arterioles, the trophic role of which becomes preponderant at this level.

The intracranial optic nerve and the optic chiasma are supplied by the internal carotid, the anterior cerebral, and the anterior communicating arteries.

As to the chiasma, our observations have led to the following conclusions:

(1) The only specific and well-defined artery which we found at the level of the chiasma was the chiasmal artery, which derives from the anterior communicating artery and supplies the central part of the chiasma (Fig. 2, opposite). However, this vessel, which has not been described before, was found in only one-third of our cases.

(2) The only characteristic arteriolar formation is the prechiasmal arch over 180°.

(3) The branches of the internal carotid artery reach the chiasma at its inferior temporal surface.

(4) The branches of the anterior communicating and the anterior cerebral arteries reach the chiasma at its antero-superior surface.

(5) Anastomoses can be observed between the small arterioles at the surface of the chiasma.

(6) All these arterioles proceed to the chiasma, either directly or—more frequently—by way of the prechiasmal arch.

(7) The ophthalmic, anterior choroidal, middle cerebral, and posterior communicating arteries are only slightly and at best indirectly involved in the vascularization of the chiasma.

The optic tract is likewise supplied by the posterior communicating artery as well as by the anterior choroidal artery.

The surface of the external geniculate body is covered by a dense anastomotic network; this is formed by branches of the anterior choroidal, and especially by branches of the posterior cerebral artery (posterior choroidal arteries) and sometimes also by a branch of the superior cerebellar artery. From this superficial network arise arterioles which penetrate obliquely the subjacent organ.

A comparison of the different parts of the primary optic pathways leads to the following conclusions:

(1) At two ends—at the level of the intrabulbar and juxtabulbar part of the optic nerve and at the level of the external geniculate body—the vascularization becomes singularly important: in front are the central retinal artery, the anterior branch of the central artery of the optic nerve, and the ciliary arteries, and behind are the posterior cerebral and anterior choroidal arteries and the branches of the vascular system of the optic tract.

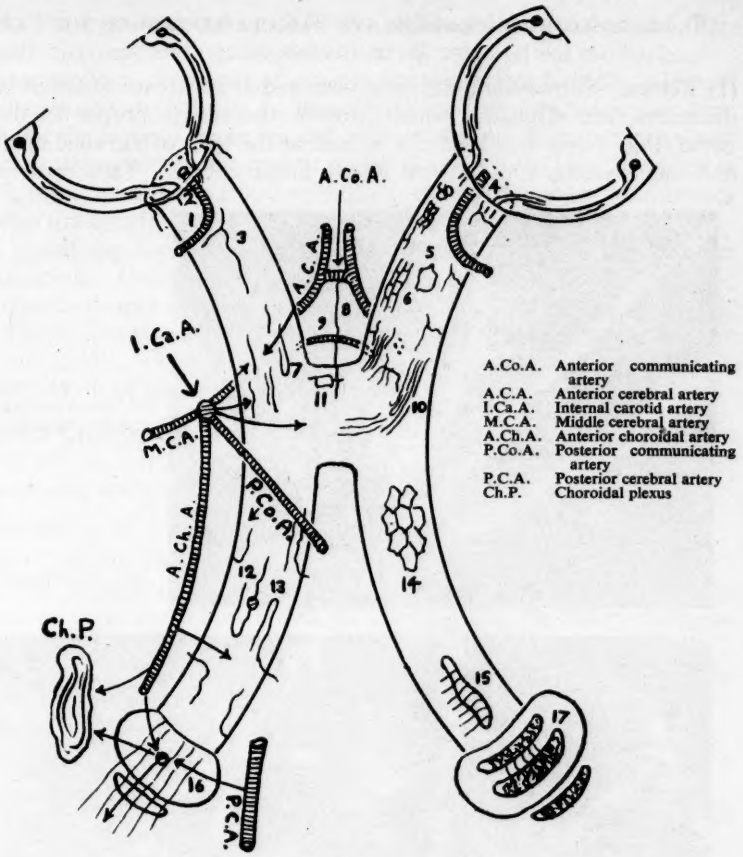


FIG. 2.

1 Central retinal artery
2 Collateral vessels of central retinal artery supplying only the retina
3 Central optic nerve artery (anterior and posterior branch)
4 Capillaries and precapillaries of optic origin localized in fascicular layer of retina
5 Specific capillaries of optic nerve: transverse units of pentagonal shape
6 Specific capillaries of optic nerve: longitudinal units of square shape
7 Nasal side of intracranial optic nerve: precapillaries curving back in hairpin bends
8 Chiasmal artery
9 Prechiasmal arcade
10 Temporal side of chiasma: capillaries curving back in hairpin bends
11 Capillary junction pattern of middle part of chiasma
12 Capillaries of optic tract arising from perforating arteries
13 Intra-tissular arterioles of optic tract with hairpin curves
14 Specific capillary structure of optic tract: polygonal, often hexagonal meshes
15 Quadrangular capillary structure of optic tract towards external geniculate body
16 Perforating arterioles and arterioles supplying the external geniculate body arising from superficial anastomotic network
17 Two types of capillary of external geniculate body characteristic of cellular and fascicular layers

(2) The intra-orbital part of the optic nerve possesses an axial trophic system (central artery of the optic nerve). At the level of the optic tract there is no single axial artery; but several arterioles—extending in an antero-posterior direction and with a reduced lumen—form what may be called a bundled axial system.

(B) Microscopic (intra-tissular) Vascularization of the Primary Optic Pathways

(1) Retina.—Surrounding the optic disc, and at a distance of about two disc diameters, are capillaries which provide the vessels proper to the optic nerve (Figs 3 and 4). They curve back at the level of the edge of the disc, and then assume a radial and nearly linear course. They form a dense

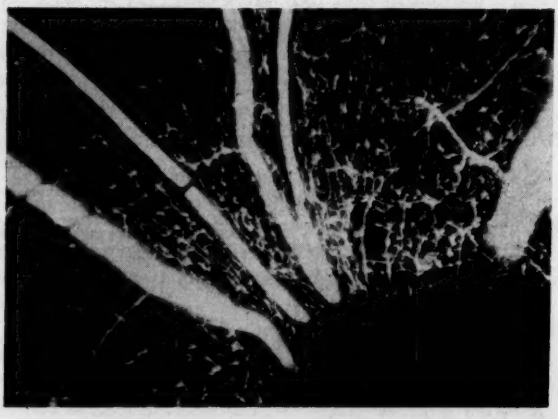
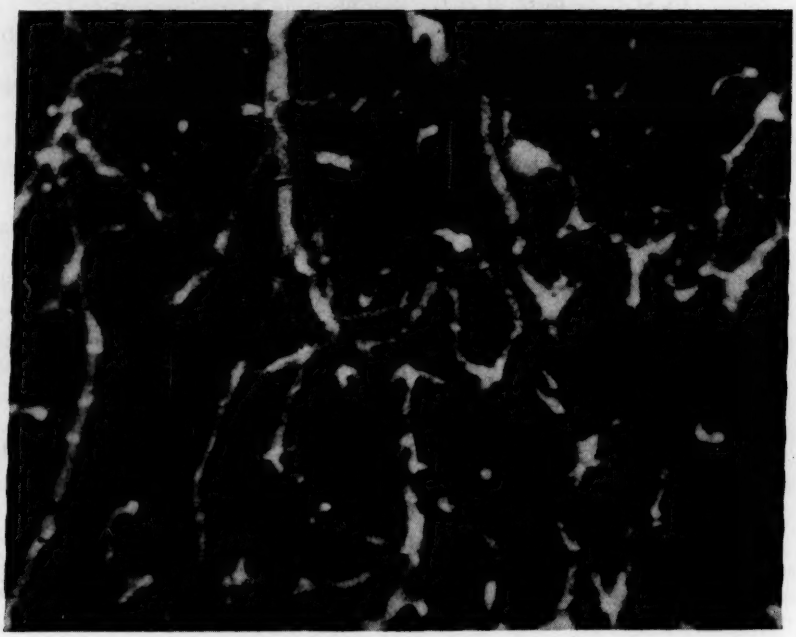


Fig. 3.—Microradiograph of retinal peripapillary capillary network. Thorotrast ×75.

Fig. 4.—Enlargement of part of Fig. 1. ×190.



network, the appearance of which is very different from the bunched, harmoniously disposed, and free appearance of the other retinal capillaries.

The peripapillary capillaries of optic origin, which may have the calibre of precapillaries and which in fact create a cilio-retinal anastomosis, are localized in the fascicular layer of the retina and anastomose with the capillaries which are directly dependent on the central retinal artery. The latter, arranged in a double layer, involve the entire membrane, but the density of the network diminishes towards the periphery.

Only round the disc does the layer of retinal nerve fibres have a proper vascularization. Otherwise there are generally only precapillary vessels; the capillaries proper are localized within and outside the layer of bipolar cells. The retinal layers localized outside the external plexiform layer are devoid of vessels, which does not necessarily mean that their supply is dependent on the choriocapillaries.

(II) Lamina Cribrosa Sclerae and Optic Disc.—The central retinal artery as a rule gives off no branch to this region. Yet the region has an abundant vascularization, the vessels originating from other sources, as follows:

(1) *Scleral*

(*a*) Capillary ramifications originating from the circle of Zinn-Haller formed by branches of the short posterior ciliary arteries. These ramifications form anastomoses with the capillaries of the anterior branch of the central artery of the optic nerve;

(*b*) Ramifications of the short posterior ciliary arteries which irrigate the sclera and then continue towards the lamina cribrosa.

(2) *Neural*

(*a*) Branches issuing from the vascular network of the pia mater;

(*b*) Ramifications of the anterior branch of the central artery of the optic nerve which anastomose with those forming the circle of Zinn-Haller.

(3) *Choroidal.*—A few recurrent branches of the choroid vessels.

(III) Intra-Orbital Part of Optic Nerve.—The intra-orbital part of the optic nerve has a characteristic capillary network, especially in its middle third, and shows two well-defined types of units:

(1) Longitudinal units running in an antero-posterior direction inside the interfascicular spaces (Fig. 5). From these units derive all the branches contributing to the formation of other vascular elements.

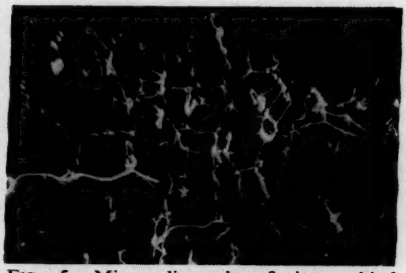


FIG. 5.—Microradiograph of intra-orbital optic nerve, middle part, longitudinal section. Thorotrast × 50.

(2) Transverse units of pentagonal and sometimes circular shape surrounding each bundle of nerves and forming complete or incomplete rings which are found at regular intervals throughout the length of the bundle (*Fig. 6*).

The capillaries deriving from these two types of units form an apparently inextricable network which shows more or less square meshes in longitudinal sections.

Posteriorly, the vascularization becomes less dense (Fig. 7) and the transverse capillary units stretch to lose their pentagonal shape; anteriorly, towards the optic disc, *the capillary network* becomes denser and more irregular.

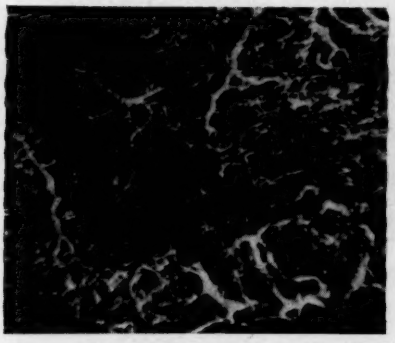


FIG. 6.—Microradiograph of intra-orbital optic nerve, middle part, transverse section. Thorotrast ×70.

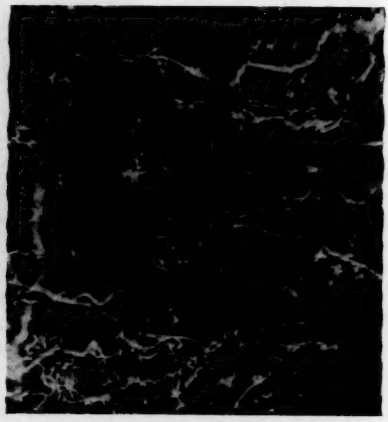


FIG. 7.—Microradiograph of intra-orbital optic nerve, anterior part, longitudinal section. Thorotrast ×70.

(IV) Intracanalicular and Intracranial Part of Optic Nerve.—These parts differ from the intra-orbital part of the optic nerve in that there is no axial artery but only precapillaries, which are less numerous than at the level of the optic tract.

The capillary network shows the following characteristics:

FIG. 8.—Microradiograph of intracranial optic nerve, longitudinal section. Thorotrast ×75.

(*a*) The transverse capillaries are stretched in an antero-posterior direction instead of remaining in the same frontal plane.

(*b*) There is no systematization. The distribution of the capillaries is irregular; considerable disorder exists both in their direction and in their mode of division (Fig. 8); angular forms are frequent.

(c) On the temporal side the capillaries, and on the nasal side especially the precapillaries, sometimes curve back in hairpin fashion; this phenomenon is also seen in the arterioles, and more frequently at the level of the optic tract.

(V) **Chiasma.**—Seven different zones can be distinguished (Fig. 9).

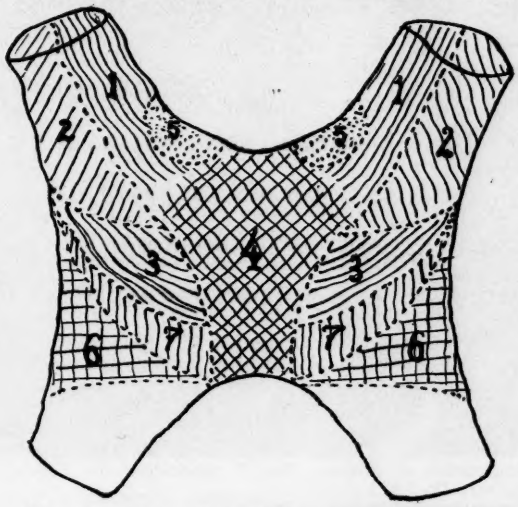


FIG. 9.—The seven zones of the chiasma (François, Neetens, and Collette, 1956b, Fig. 2, p. 733).

(1) A zone occupying the nasal half of the optic nerve and the anterior part of the chiasma, showing fine and irregularly rectilinear capillaries following the direction of the fibres and becoming more irregular in the chiasma proper (Fig. 10, overleaf). These are the longitudinal units of the optic nerve which are prolonged into the chiasma, where they curve back towards the median plane, describing a loop with an anterior concavity. The hairpin curves are less numerous here than in Zone 2.

(2) A zone occupying the temporal half of the optic nerve and the anterior part of the chiasma, showing capillaries slightly larger than those in Zone 1 (Fig. 11, overleaf). These capillaries run in all directions and make an irregular angular network.

(3) A third zone, continuing from the preceding and extending to the sagittal plane, showing finer and more regular capillaries, reminiscent of those in Zone 1. The capillaries of the temporal part of the optic nerve do not continue to the optic tract but curve back to the sagittal plane after having penetrated the chiasma.

(4) The median zone, more or less hexagonal, extends from the anterior border to the posterior border of the chiasma. Its capillaries are larger and run in a transverse direction, connecting the left and right sides. These are the junction capillaries (Fig. 12, overleaf) from which finer capillaries arise. The capillary network is less dense and less tortuous in the central part than

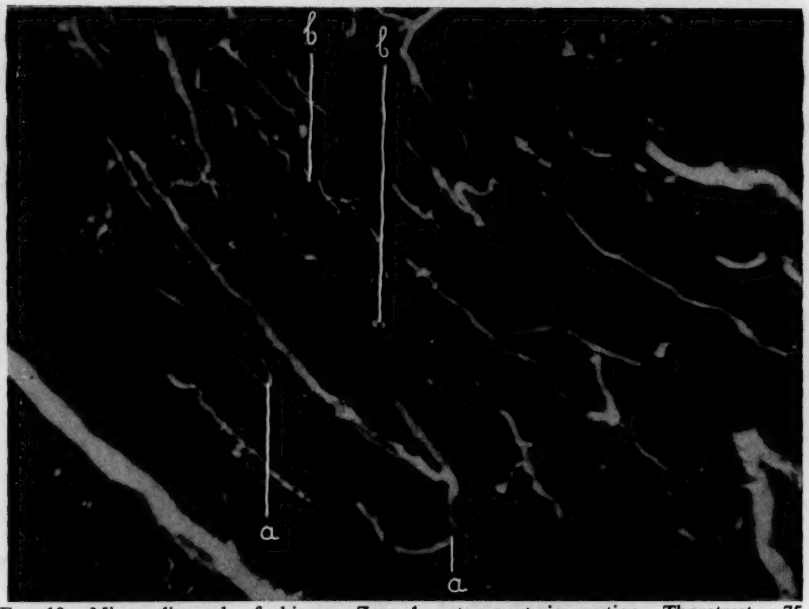


FIG. 10.—Microradiograph of chiasma, Zone 1, antero-posterior section. Thorotrast ×75. (François and others, 1956b, Fig. 3, p. 733).

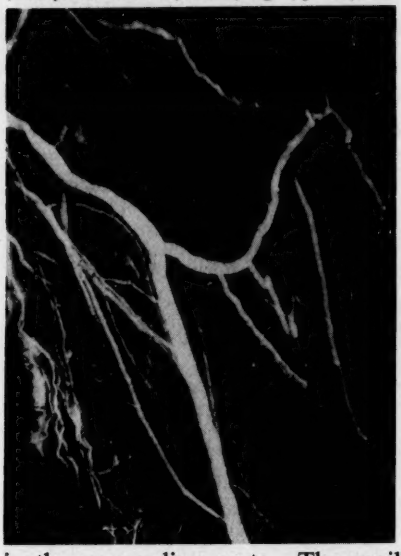


FIG. 11.—Microradiograph of chiasma, Zone 2, antero-posterior section. Thorotrast ×52·5.

in the paramedian parts. The capillaries generally follow the track of the fibres. This is the only chiasmal zone with characteristic capillaries which form true meshes and show a regular aspect throughout.

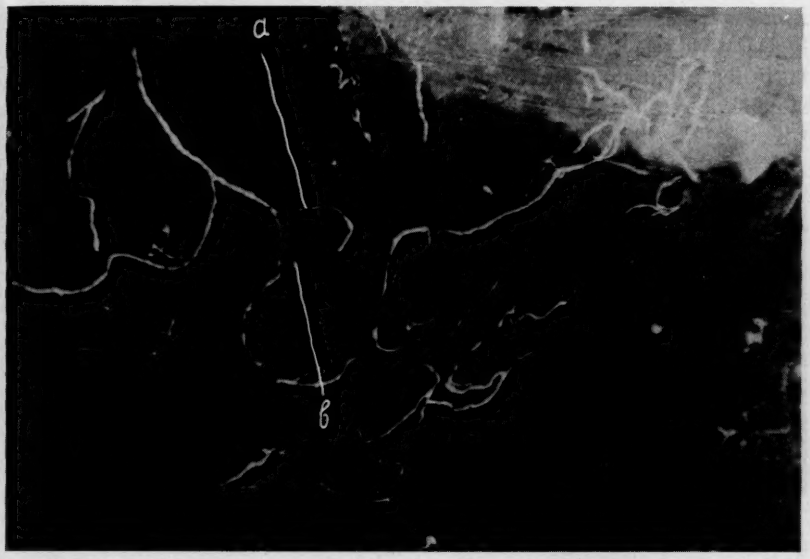


FIG. 12.—Microradiograph of chiasma, Zone 4, antero-posterior section. Thorotrast ×75. (François and others, 1956b, Fig. 7, p. 736).

(5) In the transitional region between the optic nerve and the chiasma, there exists, on the nasal side, a zone in which the capillaries are fairly fine, extremely irregular, and very confused. Capillaries with no well-defined directional pattern show abrupt changes in direction with rounded angles.

(6) The sixth zone consists of a triangular region, with its base on the temporal border of the chiasma and directed towards the posterior border. Fairly important capillaries are found here and those on the temporal side especially are closely similar to the capillaries of the optic tract (Fig. 13).

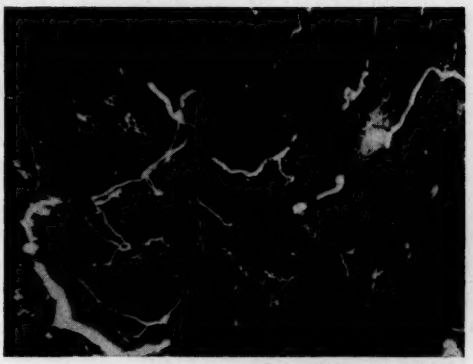


FIG. 13.—Microradiograph of chiasma, Zone 6, antero-posterior section. Thorotrast ×52·5.

(7) The last zone is one of transition between the third and the sixth

zones. It has the form of a half-moon and stretches from the temporal border of the chiasma to the median zone. It includes tortuous capillaries, fairly large but not numerous, which form a network with no apparent systematic arrangement and no well-defined structure (Fig. 14).

FIG. 14.—Microradiograph of chiasma, Zone 7, antero-posterior section. Thorotrast ×75.

The significance of these different zones is not known, but there is no congruence between the myelo- and the angio-architecture.

(VI) Optic Tract.—Longitudinal sections show the following structures (Figs 15 and 16):

FIG. 15.—Microradiograph of optic tract, longitudinal section. Thorotrast ×52·5.

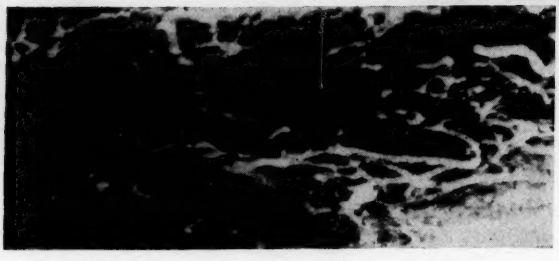


FIG. 16.—Microradiograph of optic tract, longitudinal section. Thorotrast ×195.

(1) Large and irregularly polygonal meshes which are often hexagonal.

(2) These meshes alternating with other meshes which are straighter, more elongated, and more fusiform.

(3) At the level of certain points of intersection a capillary is seen to take a circular or ellipsoid course, and to give rise to two transverse and two longitudinal capillaries; this condition is characteristic of the optic tract.

Transverse sections show the following arrangements:

(1) The capillaries never form a complete ring round the bundles of nerves.

(2) Anastomotic capillaries are localized in an oblique direction.

(VII) External Geniculate Body.—The capillary network of the external geniculate body, which anastomoses with other capillary networks, constitutes a more or less spherical formation, readily recognizable by its extreme abundance. It is composed of several layers of capillaries showing two distinct alternating types (Figs 17 and 18, overleaf):

(1) Capillaries which form extremely close meshes, round or slightly ellipsoid, and arranged in a cluster, which are typical of the cellular layers of the geniculate body; each group of nerve cells would seem to have its cluster of capillaries.

(2) Capillaries which are more linear, more widely spaced, and slightly larger, and partly arise from the optic tract; they are peculiar to the fascicular layers where they form a network similar to that of the optic tract.

(C) Comparison between the Retina and the External Geniculate Body

A comparison between the retina—starting point of the first neuron of the optic pathways—and the external geniculate body—the first relay—shows the following points of similarity:

(1) The receptor part of the retina, localized outside the external plexiform layer, is avascular; this part has no corresponding part at the level of the external geniculate body.

(2) The bipolar and ganglion cells of the retina correspond to the cellular layers of the external geniculate body, while the layer of retinal optic fibres corresponds to the fascicular layers of the ganglionic relay.

As regards vascularization, too, there are several similarities between the retina and the external geniculate body:

(1) The peripapillary capillaries, dependent on the vessels belonging to the optic nerve and occupying the fascicular layer of the retina, are analogous to those of the fascicular layers of the external geniculate body, which partly arise from the optic tract.

(2) In both organs the fascicular layers are relatively poorly vascularized, whereas the vascularization of the cellular layers is abundant.

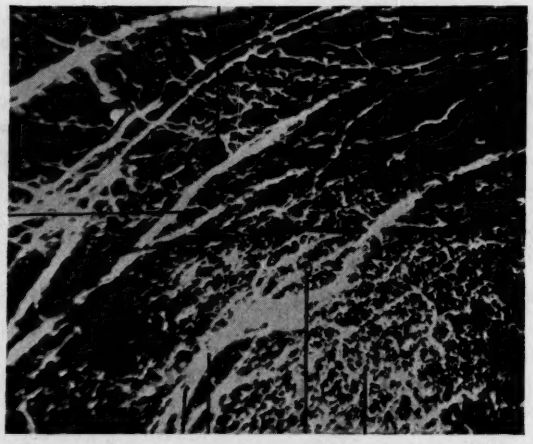


FIG. 17.—Microradiograph of lateral geniculate body, parasagittal section. Thorotrast ×75. (François and others, 1956a, Fig. 10, p. 350).

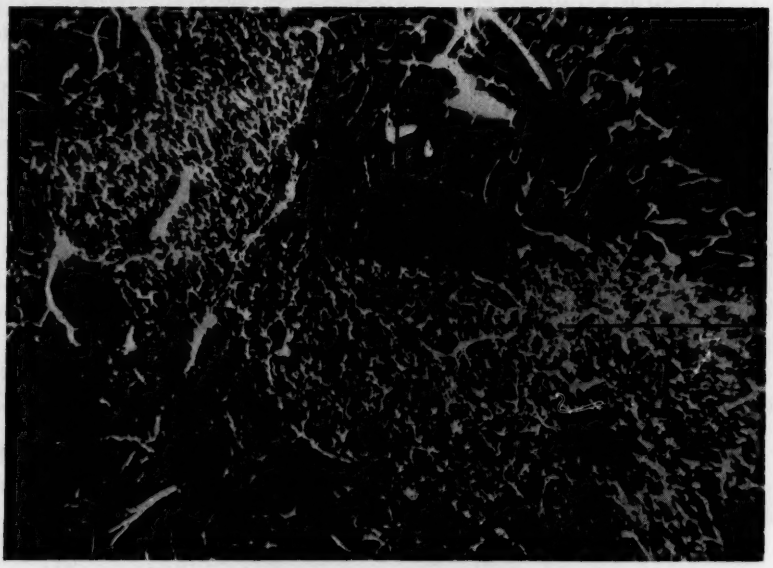


FIG. 18.—Microradiograph of lateral geniculate body, parasagittal section. Thorotrast ×75. (François and others, 1956a, Fig. 13, p. 352).

(3) The ellipsoid or round meshes formed by the capillaries are found both in the retina and in the external geniculate body, where they are smaller and more tightly packed.

(4) The vascular branches extending to a certain part of the organ remain individual and isolated both in the retina and in the ganglionic relay.

The numerous differences which appear are more apparent than real:

(1) The retina is supplied by a single specific artery (the central retinal artery). The external geniculate body depends on an anastomotic network formed by several arteries.

(2) At the level of the retina the capillaries form a double loop round the bipolar cells; in the external geniculate body nearly every cell or group of cells is surrounded by capillaries.

(3) The branches of the central retinal artery do not extend to other regions, but are undoubtedly terminal; at the level of the external geniculate body there are perforating arteries which in their course give off collaterals.

These differences are due to the two following facts:

(1) The retina constitutes an extremely thin membrane, not exceeding 0·2–0·3 mm. in thickness; it is therefore practically two-dimensional. The geniculate body, however, is definitely three-dimensional, and thus requires an enrichment of the network either by arterioles or by capillaries.

(2) The retina is the starting-point of the optic pathways, whereas the external geniculate body is merely a relay. This explains why the latter depends on various non-terminal arteries, as is the case with all ganglionic relays.

Despite apparent differences, therefore, it can be said that the structure of the retina is identical with the architecture of the external geniculate body. The vascularization of the geniculate body does in fact show the spatial construction of the retinal vascularization.

These capillary findings afford corroborative evidence of the interrelationship between the retina and the brain—a fact long established in embryology and anatomy.

(D) Comparison between the Intra-Orbital Part of the Optic Nerve and the Optic Tract

There are numerous discrepancies between the vascularization of the intra-orbital part of the optic nerve and that of the optic tract:

(1) In the intra-orbital part of the optic nerve there exists a specific axial vascular system represented by the central artery of the optic nerve. No single axial system exists at the level of the optic tract.

(2) The arterioles penetrate the interior of the optic nerve following a course perpendicular to its surface. They form branches to the nerve axis which maintain their original direction, whether this be anterior or posterior. The arterioles penetrate the interior of the optic tract following an oblique, slightly sloping course. They form branches which curve back in hairpin fashion, completely changing their direction. Since these arterioles depend on two different arteries (the anterior choroidal and the posterior communicating artery), it can be said that a double vascularization exists at the level of the optic tract which is not found at the level of the optic nerve.

(3) The capillary network of the optic nerve shows a structure completely different from that of the optic tract (Table I). Briefly, the typical aspect of the capillary network of the optic nerve shown in transverse sections is roughly repeated at the level of the optic tract, but in longitudinal sections. In the same manner, the quadrangular capillary meshes of the optic nerve seen in longitudinal sections are more or less repeated at the level of the optic tract, but in transverse sections.

TABLE I

Structure	Optic Nerve	Optic Tract
Longitudinal Capillary Unit	Follows a linear course and constitutes the basic structure	Does not exist
Transverse Capillary Unit	Specifically pentagonal in shape	Is partly lacking
Longitudinal Section	Quadrangular meshes	Sometimes fusiform and sometimes polygonal meshes, often hexagonal; characteristic of the tract
Transverse Section	Pentagonal meshes, characteristic of the optic nerve	No characteristic meshes

The continuity of the capillary network is less systematized and more irregular at the level of the optic tract than at the level of the optic nerve. No long antero-posterior capillaries are found, the longitudinal continuity being ensured by transverse capillaries.

(4) The structure of the optic nerve resembles that of the peripheral nerves whereas that of the optic tract approaches that of the white matter of the brain.

There are therefore also certain analogies between the two organs, in that whereas the capillaries of the optic tract continue towards the fascicular layers of the external geniculate body, those of the optic nerve are prolonged in the fascicular layer of the retina.

(E) General Considerations

A change in the nutrition of an organ depends in general on an alteration of the arteries, arterioles, precapillaries, or capillaries. The cause of this alteration may be found outside the vessel or within it.

Extravascular causes include intra-tissular proliferative or oedematous processes which flatten the vessels, processes of compression which concern the organ itself or its nutritive vessels, and the cicatricial processes which produce adhesions with retraction as in optochiasmal arachnoiditis. Intravascular causes include arteriosclerosis, spasm, endarteritis, embolism, etc.

The principal characteristics of the vascularization of the primary optic pathways are presented in Table II (opposite).

TABLE II

Organ	I. Retina		II. Optic Nerve				III. Chiasma	IV. Optic Tract	V. Geniculate Body	
			Intra-bulbar	Intra-orbital	Intra-canali-cular	Intra-cranial				
Function ..	Recep-tion	Con-duc-tion	Con-duc-tion	Con-duc-tion	Con-duc-tion	Con-duc-tion	Conduc-tion	Conduc-tion	Recep-tion	Conduc-tion
Arteries and Arterioles ..	–	+	+ +	+	+	+ +	+ + +	+ +	+ +	+
Precapillaries and Capillaries ..	+ + +	+	+ + +	+ +	+ +	+ +	+ +	+ +	+ + +	+
Type	Terminal		Ana-sto-moses	Central part termi-nal Peri-pheral part anasto-motic	Anastomoses		Possibility of anas-tomoses	Anasto-moses Double vascu-lariza-tion	More or less ter-minal	Possibility of anas-tomoses

CONCLUSIONS

(1) We have seen that the longitudinal capillary network of the optic nerve is normally continued towards the retina to form there a peripapillary network, which anastomoses with the capillary network proper of the retina. In short, this peripapillary network, which originates either from the circulation of the pia mater, or from the ciliary system, or from the circle of Zinn-Haller, constitutes a kind of cilioretinal vascularization. It explains the atrophic rings that are found around the disc in certain degenerative or atrophic affections of the optic nerve.

(2) The capillary and arteriolar vascularization is denser in and around the disc than in the retina or in the optic nerve itself. This explains the frequency and intensity of papilloedema.

The richness of vessels in the disc, as well as the independence of the central retinal artery during its course through the optic nerve, is also of great significance for the development and pathogenesis of functional disturbances in glaucoma.

(3) Arteriosclerosis may produce lesions not only in the optic nerve, but also in other parts of the optic pathways, either by obliteration of a nutritive artery, which, moreover, may compress the nerve fibres, or by obstruction of a capillary network of greater or lesser significance.

Possible interruption of perioptic arterioles has less effect if there are many of these arterioles (for instance, in the chiasma) or if there are numerous anastomoses (as in the peripheral network of the optic nerve).

However, if the interruption takes place in a more or less isolated or partially terminal artery, such as the central artery of the optic nerve, an acute syndrome is seen to develop, with significant functional signs. Besides

"infectious or inflammatory" papillitides or optic neuritides, there must exist a papillitis or a "vascular" neuritis, due to spasm or partial or total thrombosis of the central artery of the optic nerve, and comparable to embolism of the central retinal artery. This vascular papillitis, which may be observed, for example, in temporal arteritis, is characterized by oedema or atrophy of the disc with diminution of central vision and perimetrical defects, but possibly with preservation of the peripheral areas of the visual field, since the peripheral nutritive vessels of the optic nerve may remain intact.

(4) In the intracerebral portion of the optic nerve, the precapillary and capillary vascularization is not only irregular but also less dense temporally than nasally, so that the temporal part of the optic nerve is more fragile than the nasal part; certain defects of the nasal area of the field of vision are certainly better explained by inhibition or arrest of the capillary or precapillary circulation than by direct pressure by the sclerotic carotid arteries, the more so as the latter, if they are not rigid, are more readily deformed than the nerve itself.

(5) It is inconceivable that the chiasma, which is profusely supplied by eleven arteries and a whole network of anastomotic arterioles (a prechiasmal arteriolar arcade), should be functionally impaired by the occlusion of one or even more arteries. The functional defects should be attributed to an intra-tissular capillary affection due either to degeneration, or, more frequently, to compression from outside.

(6) In regard to the optic tract, various authors have noticed with astonishment the absence of homonymous hemianopia after ligation of the anterior choroidal artery in order to suppress the action of the corpus striatum (corpus of Luys) in certain cases of extrapyramidal trembling. The absence of this hemianopia can be explained by double vascularization (hairpin curves of the precapillaries originating either from the anterior choroidal artery or from the posterior communicating artery).

However, this hemianopia sometimes exists in cases of ligation of the anterior choroidal artery, since anomalies of the circle of Willis are frequent; in the same way, we also observed complete or partial absence of the posterior communicating artery.

(7) Since the capillary network in the lateral geniculate body, as in the retina and the optic disc, is very dense, it must frequently show oedema, but that remains an unexplored region clinically as well as experimentally.

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CAPILLARY-GLIA RELATIONSHIPS IN THE HUMAN OPTIC NERVE, CHIASMA, AND TRACT, AS SHOWN BY PHASE-COLOUR CONTRAST AND MICRODISSECTION*

BY

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IN the optic nerve, chiasma, and tract the component most closely associated with the blood vessels is the neuroglia. It has become increasingly appreciated that in the brain and spinal cord the neuroglia has a dynamic role to play in metabolic processes, and is no longer to be regarded as primarily a supporting matrix; on the details of its association with the vascular system there is little agreement. As an extension of the central nervous system, the complex glial system in the optic nerve, chiasma, and tract can be expected to play an equally important part in function and pathology.

In view of the importance of the relationship between the vascular system, especially the capillaries, and the neuroglia and the metabolic activity of the neural elements in the central nervous system, an investigation was carried out by new methods to determine the distribution and relationships of the perivascular glia in normal human optic nerve, chiasma, and tract. The normal capillary-glia architecture in different regions of the optic system must be taken into account in instances of visual field defects which arise as the result of vascular disturbance or some other pathological condition. It is perhaps in the capillary-glia architecture that we might seek an explanation of certain differences in visual field defects which result from disturbances in the vascular system.

The literature on the glia of the optic system is remarkably scarce. Liss (1956) remarked that he could find only three reports on the astroglia in the optic nerve (Marchesani, 1926; Cone and Macmillan, 1932; Kolmer, cited by Lauber, 1936).

The astroglia are the elements of the neuroglia which have special relationships to the vascular system. Cone and Macmillan (1932) cited Marchesani's description of the astroglia in the optic nerve as shown by staining methods, and mentioned that the glia intervened in a typical manner between the vascular and nervous systems at the lamina scleralis. This followed the view of Achucarro (1918), who regarded this relationship as characteristic of the whole central nervous system. Cone and Macmillan (1932) stated that the neuroglial cells in the optic system did not differ in any way from those in the brain and spinal cord.

* Read at the combined meeting of the Society of British Neurological Surgeons and the 36th Annual Meeting of the Irish Ophthalmological Society in Dublin, May 16–18, 1957.

Among the questions raised by a review of investigations on the blood vessels of the cortices of the brain and central tracts and the neuroglia are those which concern the existence of the glia membrane around the capillary endothelium, and if present its character and structure. How close is the relationship of the membrane or the glia cells and their processes to the capillary wall, and to the arteriole and venule adventitia? Does the glia differ in its degree of attachment to these different vessels in the nerve, chiasma, and tract? Can a potential space be demonstrated between the vessel wall and glial structures (the so-called space of His)? Further, does a subarachnoid extension (Virchow-Robin space) pass as far as the capillaries in the nerve, chiasma, and tract and thus intervene between the nutritive capillary and nerve elements? At least some of these questions may be answered by newer approaches to the problem of the glia vascular relationships.

Material and Methods

The material consisted of eight specimens of intracranial optic nerve, chiasma, and part of the optic tract behind the chiasma. These were obtained within 8 hours of death, chiefly in cases where this occurred suddenly by accident. In two instances the specimens were obtained in less than 2 hours in favourable conditions.

Frozen and paraffin sections were cut from the 48-hour formalin-fixed and brom-formalin-fixed specimens. The sections varied in thickness from 1 to 5 μ. Control sections were stained with Cajal gold sublimate, del Rio Hortego silver carbonate, gallocyanin, and acid fuchsin. The sections for phase contrast-colour contrast microscopy were mounted unstained in glycerin jelly and glycerin and distilled water on slides and on cover slips. Examination of the sections was carried out with a new Leitz phase contrast-colour contrast condenser. Microdissection of the specimens was performed with a large sliding micromanipulator under optical phase and colouration. The instruments used were glass needles drawn to a tip diameter of approximately 8 and 20 μ.

The specimens were photographed during the operations, using Anscochrome 32 ASA film rated at 64 ASA and 100 ASA, and were developed accordingly.

Normal Vascular-Glia Anatomy under Phase-Colour Contrast

Attention was mainly directed to the capillary-glia relationships. The most favourable specimens were those fixed within 2 hours of death; changes in the cytological detail could be detected at 8 hours. All the illustrations reproduced below are taken from original 35-mm. colour transparencies.

Optic Nerve.—The relations of the capillaries contained in the pial septa were easily recognized (Fig. 1, opposite). The septa were seen to be covered by a neuroglial meshwork and vascular footplates. Capillaries between the nerve bundles which completely lack a connective-tissue covering could not be definitely identified. A glial meshwork was also clearly visible outside this thin connective tissue around an isolated capillary (Fig. 2, opposite).

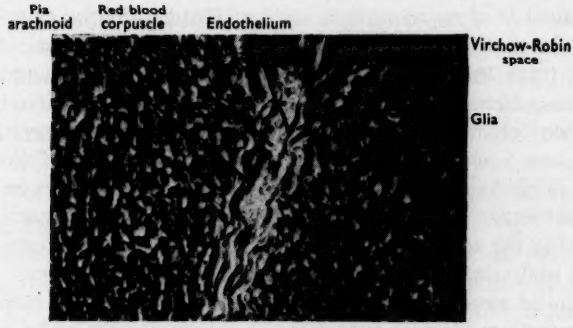


FIG. 1.—Optic nerve: pia-arachnoid extension in septal region; venous capillary visible in centre of connective tissue. ×400.

FIG. 2.—Optic nerve: segment of capillary with pia-arachnoid connective tissue between endothelium and glia. ×480.

Chiasma and Tract.—In general the architecture of the chiasma is distinctive and different from that of the optic nerve. In particular most capillaries do not possess the pial covering which was seen to be characteristic of the nerve (Fig. 3). A glial mesh and vascular footplates can be

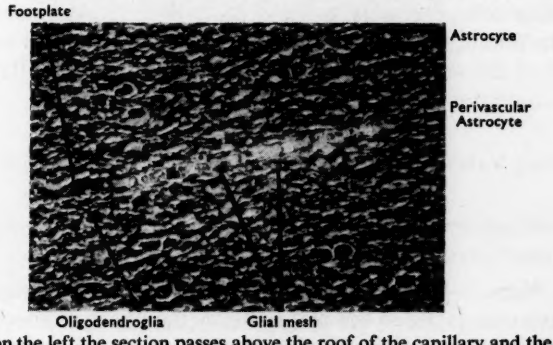


FIG. 3.—Chiasma: on the left the section passes above the roof of the capillary and the glial mesh and footplates are visible; sections of astrocyte footplates are also seen. Perivascular astrocytes are seen on the right. ×400.

distinguished in close relation to the smallest capillary. Satellite astrocytes and oligodendroglia cells could also be seen in characteristic dispositions.

In the tract the astrocytes (mainly fibrous in type according to their nuclear characteristics) and oligodendroglia were distinctive in their columnar arrangement characteristic of central tracts. Since the sections were thin (usually 2 or 5 μ) for phase, the processes could not be followed as far as those in thick-stained sections. The microglia could also be distinguished and in particular they did not show any pathological reactions; this was confirmed in the stained sections. As in the chiasma, the capillaries did not have the pial connective-tissue covering found in the nerve. These capillaries showed associated astrocytes and oligodendroglia, often arranged in groups as well as vascular footplates, in relation to the endothelium (Fig. 4).

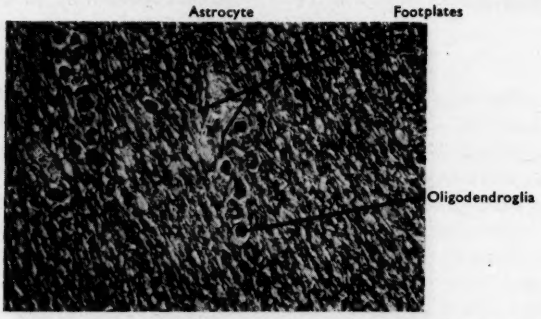


FIG. 4.—Optic tract: columnar and grouped astrocytes and oligodendroglia seen near capillary, and vascular footplates with fibrous processes on sides of endothelia. ×400.

The continuity of the glia as a membrane was in doubt. Around the larger blood vessels the space of Held was easily distinguishable with the same characteristics as were revealed by stained sections; the smaller capillaries did not show a space of Held.

The phase-colour contrast method was a useful supplement to the classical staining techniques, since it simultaneously revealed cellular and architectural details of all the structures present. A disadvantage was the fact that thick sections could not be used.

NORMAL VASCULAR-GLIA ANATOMY BY MICRODISSECTION UNDER PHASE-COLOUR CONTRAST

Care was taken to obviate artefacts in dissection by freeing the part of the section under examination from the slide before dissection.

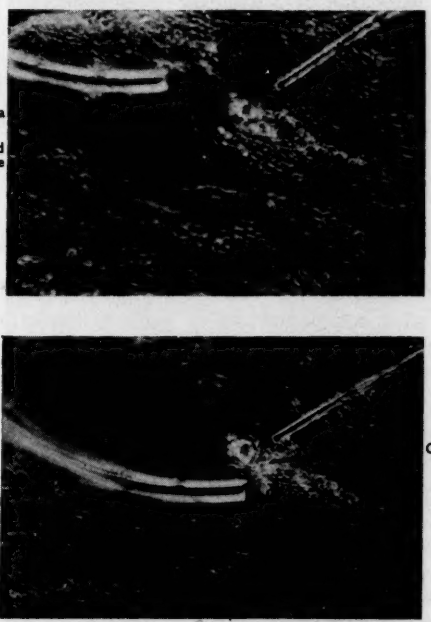
Optic Nerve.—Since an apparent Virchow-Robin space was present around the vessels inside the pial septa by the phase method, a dissection of this space was carried out to determine its actual presence. Dissection revealed that the connective-tissue layers which bordered the space were not

adherent to one another; it could be demonstrated that a Virchow-Robin space existed around the small vessels of capillary dimension in the septa.

The vessels outside the septa could not easily be freed from their connective tissue. Vascular footplates and a glial mesh were adherent to this perivascular connective tissue. Here a Virchow-Robin space could not be shown. This suggests that the Virchow-Robin space is closed and does not communicate with the potential space of His (as originally defined) around the capillaries.

Chiasma and Tract

(*a*) *Precapillaries and Arterioles.*—In posterior parts of the chiasma, where precapillaries and arterioles of about 40μ diameter can be found, it may be seen by phase-colour contrast that pial connective tissue is not present and perivascular glial footplates attach directly to the adventitia wall. By dissection, cross-section segments of these vessels could be freed and carried outside the nerve for examination (Figs 5 and 6, and Fig. 7, overleaf).



FIGS 5 and 6.—Chiasma: microdissection of precapillary; glial fibres adhere to vessel wall when segment is removed to exterior of section. ×240.

The glial mesh was stripped from the vessel segment (Fig. 8, overleaf); this manipulation does not detach at least some of the vascular footplates from the adventitia. On cross-section they cannot be seen, but on turning the section on edge the remains become visible.

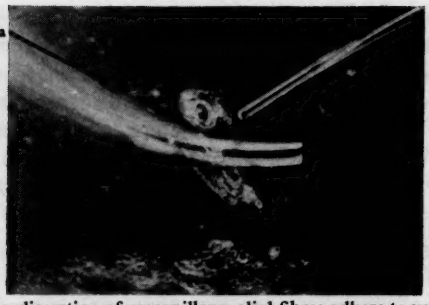


FIG. 7.—Chiasma: microdissection of precapillary; glial fibres adhere to vessel wall when segment is removed to exterior of section. ×240.

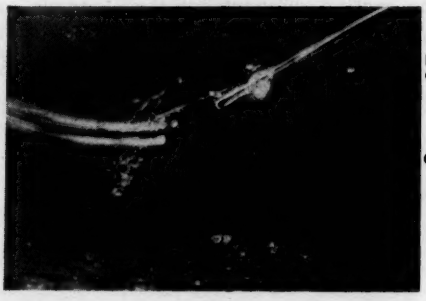


FIG. 8.—Chiasma: glial mesh stripped from precapillary wall (same vessel as in Figs 5, 6, 7). ×240.

On a longitudinal section of a vessel of capillary dimensions which showed connective tissue as a component of its wall, a dissection demonstrated a similarly adherent glial meshwork (Fig. 9, and Figs 10, 11, 12, opposite).

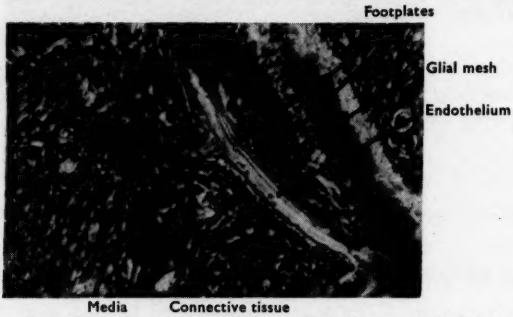
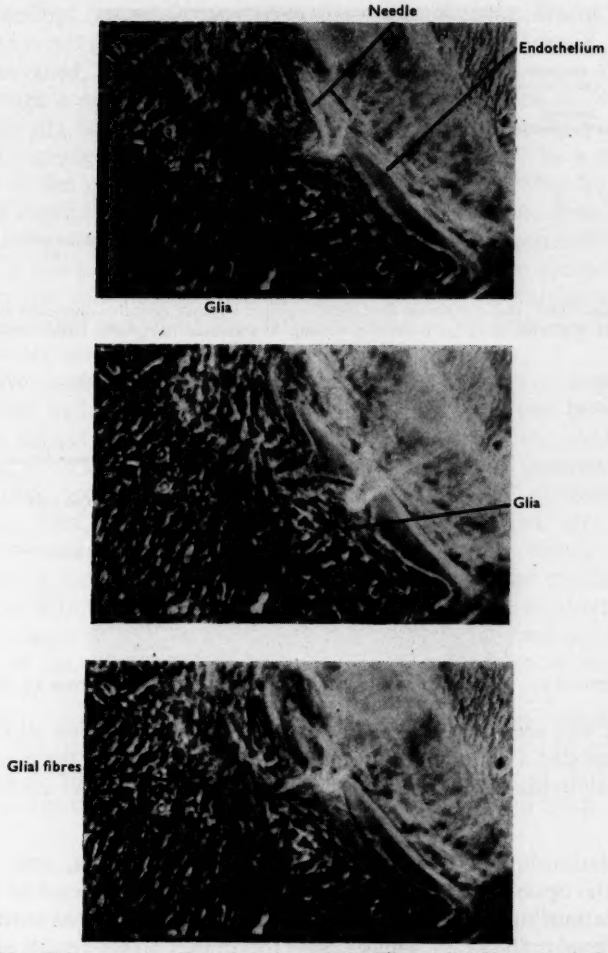


FIG. 9.—Chiasma: microdissection of arterial capillary; glial fibres attached to capillary wall remain adherent and resist stretching of wall. Diameter of needle-tip 8μ. ×400.

(*b*) *Capillaries.*—The capillary-glia relationships were examined by microdissection in the absence of a pial covering or connective tissue in the capillary wall (Fig. 13, overleaf).



FIGS 10, 11, 12.—Chiasma: microdissection of arterial capillary; glial fibres attached to capillary wall remain adherent and resist stretching of wall. Diameter of needle-tip 8μ. ×400.

On a longitudinal section, the capillary-glia network may be dissected to determine the degree of attachment of the glia and its structure. This was done by drawing the glia away from the endothelium, where it was seen that the two structures did not adhere to one another.

This procedure demonstrated a space of His (Fig. 14, overleaf); the glia was shown to consist of a fine mesh of sponge. At the capillary wall the mesh was seen to be bordered at least partly by a cuticle or membrane which was not apparent before dissection.

FIG. 13.—Optic tract: two capillaries seen close together without definite connective tissue outside endothelium; segments of glial processes ending in vascular footplates make connexion with endothelium. ×450.

FIG. 14.—Optic tract: glia drawn away from endothelium and artefact space of His produced (same section as in Fig. 13). ×450.

Thus it was seen that the glia was more closely attached to the vessels which possessed a definite adventitia and to pial connective tissue around the vessels than to the capillary where no connective-tissue layer could be seen.

DISCUSSION

The relationship between the capillaries, the neuroglia, and the nerve tissue in the optic nerve, chiasma, and tract, might be expected to be similar to the relationships which are found in the brain and spinal cord. At the capillary level in the brain, Patek (1944) found that on the results of injection experiments no barrier between the vascular system and the nerve tissue existed. However, Glees (1955) found a definite delay in the transmission of injected dyes which must have its origin at capillary level, a finding supported by studies with radioactive isotopes. The situation in the optic nerve, chiasma, and tract was not discussed by Glees.

A close connexion between the oligodendroglia, astrocytes and their footplates, and the capillary wall has been assumed on the basis of staining methods. By microdissection under phase-colour contrast, it has been possible to demonstrate directly that the degree of attachment of the astrocytes and their processes to the blood vessels seems to depend on the presence

of an adventitial layer. If adventitia is present the attachment is fixed, whereas if it is not present a cleavage space can easily be produced.

Glia intervened between the vascular and nervous tissue in the optic system, though a definite continuous membrane could not be shown in this instance; the glia intermediary consisted, apart from vascular footplates, of a fine mesh or sponge of glial fibres, bordered at least partly by a cuticle or membrane at the capillary level not clearly shown in staining techniques. The view of Patek (1944) that the so-called space of His in the brain between the glial mesh and the adventitia is erroneously described seems correct. It is doubtful if this potential space could be injected, since the glia is attached to the arteriole and venous adventitia; but around the capillaries where adventitia is absent and where the glial attachment is less fixed it would seem that an artefact space could be produced.

Connective tissue of pial origin intervened between the nerve elements and the capillaries in large parts of the optic nerve. Capillaries between the nerve fibres did not have a Virchow-Robin space around them. At the same time capillaries in the septal region of the optic nerve were surrounded by a Virchow-Robin space. Connective tissue was not found in most of the chiasma and tract, where the findings of François, Neetens, and Collette (1956) could be confirmed that there is a noticeably richer supply of capillaries. Here a Virchow-Robin space was not found at the capillary level (which agrees with the findings of Kuhlenbeck (1951) for the cerebral cortex). Thus the presence of a Virchow-Robin space seems to depend not so much on the size of the vessel as on the amount of pial connective tissue which is present around it.

The pattern described in the glia-vascular relationship raises further questions. Similar methods to those described could be used to investigate the changes in the glia-vascular pattern in experimental or pathological alterations in the circulation in the optic nerve, chiasma, and tract.

Summary

Microdissection of fixed tissues combined with a new phase contrast-colour contrast optical system was used to investigate the capillary-glia relationships in the normal human optic nerve, chiasma, and tract. It was concluded that glial elements in the form of a mesh separate the nerve fibres from the capillary at every point examined; the presence of a glial membrane was uncertain. The presence of connective tissue around the endothelium seemed to determine the degree of fixation of the glial mesh; the glia was attached to adventitia, whereas in the absence of connective tissue the glia was not fixed to the capillary endothelium. A Virchow-Robin space could be clearly demonstrated in the septal region of the optic nerve in relation to capillaries, but not at the capillary level in the chiasma and tract where perivascular pial connective tissue was usually absent; therefore the presence

of a Virchow-Robin space depended on the amount of related connective tissue and not on the size of the vessel.

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OPTIC DISC OEDEMA IN THE ABSENCE OF RAISED INTRACRANIAL PRESSURE*

BY

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PAPILLOEDEMA is of particular interest to ophthalmologists and neurosurgeons. Its pathogenesis in raised intracranial pressure due to cerebral tumour is still argued—quite apart from its explanation in other conditions. Perhaps a study of those cases, admittedly rare, in which oedema of the optic discs occurs without raised cerebrospinal fluid pressure, and of cases without papilloedema in spite of raised cerebrospinal fluid pressure, will throw light on the main problem. It is the former type of case which I wish to discuss. I had originally entitled my paper "Papilloedema in the absence of raised intracranial pressure", but I am sure that the majority of those present would prefer to restrict the term "papilloedema" to cases with raised intracranial pressure, as defined by Parsons (1908) and Paton and Holmes (1911).

In none of the cases which I am presenting was there any loss of function, such as might be associated with optic neuritis, and the intracranial pressure was proved to be within normal limits by a lumbar puncture, after preliminary investigations had determined that such a procedure was safe to carry out.

When starting work at the Midland Centre for Neurosurgery I had certain ideas about the nature and significance of papilloedema. These ideas were shaken by finding cases with appearances closely simulating papilloedema from raised intracranial pressure, in which neurological and neurosurgical investigation was essentially negative, at least as an explanation of the oedema of the disc. Five typical cases are summarized in the Table (overleaf).

Case Reports

Case 1, a man aged 60, had experienced gradual weakness of the left side of his body during the past 2 years and more recently difficulty with speech. He was very slow in all movements, to the extent of akinesia; he showed a slight weakness of the left arm and leg with increased reflexes, and a bilateral extensor plantar response. The systolic blood pressure varied between 170 and 200, and the diastolic blood pressure between 100 and 120 mm. Hg. He showed bilateral disc oedema, with increased venous pressure, more marked on the right side. The retinal arterial pressures were 110+/55 mm. Hg Right and 120+/55 mm. Hg Left. Other relevant findings were an erythrocyte sedimentation rate of 26 mm./hr; a skull *x* ray showing some osteoporosis; bilateral carotid angiograms showing only arteriosclerotic cerebral vessels. At lumbar puncture, the initial pressure was 140 mm. C.S.F., and the protein in the cerebrospinal fluid was 21 mg./100 ml.

* Read at the combined meeting of the Society of British Neurological Surgeons and the 36th Annual Meeting of the Irish Ophthalmological Society in Dublin, May 16–18, 1957.

TABLE
SUMMARY OF

Case No.	Sex	Age (yrs)	Disc Appearance	Corrected Visual Acuity		Visual Fields* Blind Spots
				Right	Left	
1	M	60	Papilloedema right > left Vein pressure up	6/5	6/6	—
2	M	66	Papilloedema right and left Later: Veins engorged right partial obstruction	6/9	6/6	Enlarged
3	M	30	Papilloedema right and left Later: Right oedema and vein engorged left normal	6/12	6/5	Right Enlarged
4	F	27	Papilloedema right and left Vein pressure response normal	6/9	6/18	Both enlarged with central spread left
5	F	48	Old Papilloedema right Left vein engorged with glia	6/6 (–4)	6/6 (–1)	Right enlarged

* Fields all

The relatively high retinal arterial pressure as compared with the systemic blood pressure, as well as the other findings, were those of hypertension passing into a malignant phase, with papilloedema and normal cerebrospinal fluid pressure.

Case 2, a man aged 66, had been diagnosed as a case of bilateral papilloedema by an ophthalmologist and a neurologist. He was admitted to hospital some weeks later, and when he was seen on February 23, 1956, the findings were less strikingly those of papilloedema and could better be described as bilateral venous engorgement, with partial obstruction of the right central retinal vein. The visual acuity was 6/9 in the right eye with glasses and 6/6 in the left eye with glasses. The visual fields were full peripherally, with enlargement of the blind spots. The pressure in the central retinal vein on the right side was 48 mm. Hg and on the left 20 mm. Hg. The ocular tension in the right eye was raised to 35 and in the left to 28 mm. Hg (Schiötz). At lumbar puncture the initial pressure was 100 mm. cerebrospinal fluid. The blood pressure was 155/110.

A few months after his return home, he was taken acutely ill with severe abdominal pain and died 2 days later on 29.9.56. A *post-mortem* examination showed haemoperi-

FIVE CASES

Dynamometry		Blood Pressure (mm. Hg)	Lumbar Puncture (Initial pressure mm. C.S.F.)	Remarks	Investigations
Right	Left				
Arterial 110+/55	120+/50	170/100	140	Akinesia Left spastic hemiparesis Plantars ↑ ↑	E.S.R. 26 mm./hr. Skull osteoporosis Angiogram arteriosclerosis C.S.F. protein 21 mg. per 100 ml.
Venous 48	20	155/110	100	Died 29.9.56 Dissecting aortic aneurysm	Ocular tension (mm. Hg Schiötz) right 35, left 28 X-ray chest and skull, right carotid angiogram normal C.S.F. protein and cells slight increase
100 Arterial 110/60	— 100/40	130/80	210	—	—
90/40	85/40	130/80	160	Later: Right disc normal left disc almost normal	C.S.F. protein—normal Cells—slight increase
120/50	130/50	145/80	110	—	X-ray chest and skull, left carotid angiogram normal Right carotid angiogram showed aneurysm of post-communicating artery

full peripherally.

toneum due to the rupture of a dissecting aneurysm of the abdominal aorta.

Case 3, a man aged 30, was referred as a case of bilateral papilloedema by an ophthalmologist, an opinion which was supported by a neurosurgeon. Clinical examination was negative, apart from the ophthalmic findings. The blood pressure was 130/80. Chest and skull *x* rays and a right carotid angiogram were normal. The cerebrospinal fluid showed a slight increase in protein and cells. Other investigations were negative.

As in Case 2, when the patient was admitted to hospital, the eye findings were less convincing, there was oedema localized to the right optic disc, with marked venous engorgement; the appearance of the left disc was now within normal limits. Visual acuity with correction was 6/12 in the right eye, and 6/5 in the left. The visual fields were full peripherally, showing an enlarged blind spot on the right side, the left being normal. The pressure in the right central retinal vein was raised to 100 mm. Hg. The left central retinal vein collapsed immediately on pressure on the globe. The right central retinal artery had a pressure of 110/60 and the left 100/40. At lumbar puncture the initial pressure was 210 mm. cerebrospinal fluid.

Cases 2 and 3 are examples of difficulty in the differential diagnosis between partial obstruction of the central retinal vein and papilloedema. Both cases show venous pressures which are sufficient to embarrass the nutrition in the region of the optic disc.

Case 4, a woman aged 27, gave a history of two attacks of headache and loss of vision, one year ago and 10 days ago, each lasting for only a short time. She was found, on examination, to have bilateral papilloedema. When seen on November 17, 1956, she still showed bilateral papilloedema, but with a normal venous pressure response; the retinal arterial pressures were 90/40 in the right eye and 85/40 in the left eye. The visual acuity in the right eye was 6/9 and in the left 6/18, with no refractive error. The visual fields showed normal peripheral isopters and enlarged blind spots with central extension towards the fixation point in the left eye. From these appearances, it seemed that the cerebrospinal fluid pressure might be normal, and at lumbar puncture the initial pressure was found to be 160 mm. C.S.F. The cerebrospinal fluid showed a slightly raised cell count, but normal protein. 2 months later (on January 1, 1957) this patient was reported to have a normal appearance of the right disc and an almost normal left disc.

It seems likely that this patient had suffered a transient rise of intracranial pressure and was admitted for investigation with oedema of the discs remaining after the cerebrospinal fluid pressure had returned to normal, as one would expect to see in benign intracranial hypertension.

Case 5, a woman aged 48, gave a history of blurring of vision in the right eye with orbital swelling. On examination in October, 1956, she was found to have right papilloedema and left venous engorgement, with glial tissue on the disc. The visual fields showed no defect on perimetry or screening. The retinal arterial pressures were 110+/50 in each eye, and the blood pressure was 145/80. The urine showed no abnormality. On February 28, 1957, the ophthalmic findings were those of old-standing papilloedema in the right eye with venous engorgement but normal pressure response; the left eye showed venous engorgement and gliosis, as previously described. The arterial diastolic pressures in the retina were 50 in the right eye and 45 in the left. The visual acuity in the right eye was 6/6 (−4) and in the left eye 6/6 (−1), with correction. The visual fields showed a full periphery, with slight contraction of the central isopters in the right eye and an enlarged blind spot. Neurological examination was otherwise negative. At lumbar puncture the initial pressure was 110 mm. C.S.F. Chest and skull *x* rays and left angiogram were normal. The right angiogram showed an aneurysm of the right posterior communicating artery.

The finding of an aneurysm in this situation was quite unexpected and difficult to relate directly to the optic disc oedema. It might lend support to the suggestion of a vaso-motor cause for the oedema of the disc, which is also supported by the high retinal arterial pressure relative to the brachial pressures.

Summary of Cases

(1) The first was a case of a hypertension passing into a malignant phase.

(2) and (3) The second and third cases were examples of incomplete central vein obstruction.

(4) The fourth was a case of papilloedema, first seen in a regressive phase.

(5) The fifth case showed a relative hypertension of cerebral circulation, with an aneurysm on the circle of Willis.

All the patients had normal cerebrospinal fluid pressures and, apart from Case 4 with probable benign intracranial hypertension in the regressive phase, they had local vascular conditions of the optic discs, produced in different ways but in each case comparable to those which occur in raised intracranial pressure.

These cases were admitted to the Midland Centre for Neurosurgery during a period when six hundred cases were admitted for intracranial conditions, eleven of these being classified as benign intracranial hypertension.

Previous Reports of Cases

One finds a great many articles entitled: "Papilloedema in a case of so-and-so", which would usually be better entitled: "Raised cerebrospinal fluid pressure in a case of so-and-so", because the histories reveal that the cerebrospinal fluid pressure was in fact raised (Watkins, Wagener, and Brown, 1941; Masson, Durix, and Perrin, 1950; Vissi and Dobrina, 1950; Devanney and Shea, 1952).

It is also clear that the term papilloedema is often used loosely to describe cases where there is considerable loss of vision or extensive field defect (Wilson and Darkes, 1933; Chamlin and Davidoff, 1950; McPherson, 1955).

It seems that in optic disc oedema associated with blood dyscrasias the cerebrospinal fluid pressure has been found to be raised whenever it has been checked, but it is conceivable that in such cases oedema might be brought about by anoxia (Watkins, Wagener, and Brown, 1941).

In hypoparathyroidism, papilloedema, when it occurs, is always associated with cerebral oedema, and in all cases where the cerebrospinal fluid pressure was measured it was found to be raised (Walsh and Murray, 1953).

Normal or low cerebrospinal fluid pressures have been recorded with disc oedema in cases of malignant hypertension, in the Guillain-Barré syndrome of polyradiculitis with high cerebrospinal fluid protein levels, and in traumatic hypotension. Mention should also be made of simulated bilateral papilloedema in association with drusen of the optic nerve.

In studying malignant hypertension, Taylor, Corcoran, and Page (1954) take the upper limit of normal cerebrospinal fluid pressure to be 250 mm., which I think will be considered a little high. However, taking this as the standard highest level, there were 100 patients who had disc oedema and 57 of these were found to have cerebrospinal fluid pressures below 250 mm. Taylor and others (1954) conclude that the association between cerebrospinal fluid pressure and disc oedema in the presence of hypertension is collateral rather than causal. The disc oedema was not associated with any evident increase in brain volume. Fishberg and Oppenheimer (1930) reached similar conclusions.

With regard to the Guillain-Barré syndrome, Drew and Magee (1951) found a total of ten cases in the literature, four of which had a low cerebrospinal fluid pressure. In their own case, there was a pressure of 150 mm. C.S.F. or less at lumbar puncture on four occasions—but 6 weeks after the onset of the disease the pressure was raised to 250 mm.

Studying ventricular hypotension, Sadoughi (1950) reported observations of cases with optic disc oedema, in which there had been no symptoms suggesting a previous hypertensive phase and the disc oedema appeared to be associated with cerebral oedema. He concluded that cerebral oedema can give rise to an oedema

of the disc, which is most frequently associated with a raised cerebrospinal fluid pressure but can be associated with a low pressure.

Discussing drusen of the optic nerve, Lyle (1953) mentioned a case which was thought to show bilateral papilloedema of moderate degree and was admitted to a neurosurgical hospital to be thoroughly investigated. The visual acuity and visual fields were normal and no abnormality was found after investigation. Chamlin and Davidoff (1950) describe cases in which nerve fibre bundle defects are shown on visual fields and serve to differentiate the condition from true papilloedema. However, Chamlin and Davidoff (1952) state that enlarged blind spots and peripheral contraction may cause the condition to resemble papilloedema very closely. Case 7 in an article on bilateral papilloedema by Abrahamson, Retter, and LaFia (1953) was that of a patient who showed drusen bodies on the right disc; in spite of this, these authors felt that the oedema which they saw was true papilloedema, because, after ventriculography, the enlarged blind spots became smaller, the patient's symptoms of headache improved, and the papilloedema subsided.

Thiébaut and Matavulj (1955) described a case of encephalitis in which hypotension was discovered at ventricular puncture. This patient had previously been given hypertonic serum to relieve her headaches. After ventricular puncture, the patient was rehydrated and then the papilloedema appeared; it therefore seems possible that the cerebrospinal fluid pressure was also raised at this time, although no estimation of the pressure was made.

Discussion

On considering the cases which I have reported and those taken from the literature, it is clear that in some conditions—namely, in partial retinal vein obstruction, in malignant hypertension, in the Guillain-Barré syndrome, and in hypotension after cranial trauma—oedema of the optic discs has been observed without a raised cerebrospinal fluid pressure. Hence, compression of the central vein as it passes across the sub-arachnoid space can be ruled out as a factor in the production of the oedema in these cases. Before discussing the manner of its production in these cases, certain facts should be noted:

Cannon (1901) pointed out that, in circumstances of non-nutrition, the brain will take up water from a solution isotonic with blood and will thereby exert pressure sufficient to exclude the blood from the cerebral vessels.

Samuels (1938) pointed out that a *post-mortem* swelling of the papilla is not unusual, and an inexperienced examiner might be induced to make a diagnosis of choked disc.

Van Heuven and Fischer (1936, 1937) showed that the brain has a water-binding capacity comparable to that of a true gel, a property shared by the optic nerve. The retina, on the contrary, swells to a much less extent; and this appears to me to be a much more satisfactory explanation of the limitation of papilloedema to the neighbourhood of the nerve head than that given by Lyle (1953), who says it is due to the firm attachment of the footplates of the fibres of Müller at the disc edge.

It seems to me that if the arterial supply to the optic nerve is cut off suddenly a complete loss of vision will result, but if it is sufficiently gradual

the reduction of nutrition and anoxia may lead to oedema without loss of function. This is a condition which may be seen in malignant hypertension. In papilloedema due to raised intracranial pressure, similar circumstances of impaired nutrition may arise at a lower arterial pressure when this is coupled with embarrassment of venous return. Finally, a similar reduction in nutrition may take place with a higher rise of venous pressure due to partial venous obstruction.

My suggestions are supported by the findings of Bailliart (1929), Sobański (1937), and Redslob (1950) that in raised intracranial tension without papilloedema the retinal arterial diastolic pressure is always found to be raised and drops rapidly on reduction of intracranial pressure. On the contrary, the retinal arterial pressure is found to fall with the appearance of papilloedema—in other words, the elevation of retinal arterial pressure is an adaptation to the intracranial pressure, and when the diastolic pressure cannot keep pace with the rise of intracranial pressure papilloedema results. A similar state of affairs in the cerebral vessels could be expected to cause cerebral oedema.

In addition, the normal ratio of arterial to venous pressure in the retina is said to be 3:1, and if this relationship holds oedema does not occur; but if the relationship reaches 3:2 oedema occurs, because the arterial pressure is insufficient to maintain an adequate capillary circulation. Support to this theory is also given by Brégeat (1956), who concluded that if papilloedema occurred in the presence of low intracranial pressure there could be no other explanation except that of vaso-motor phenomena in the optic nerve head.

Conclusion

It would appear that the type of case which I have been discussing will be encountered more frequently in the future as a result of modern methods of neurosurgical investigation. Lumbar puncture, which a few years ago might have been done as an early diagnostic procedure in such cases, will now not be done for fear of causing a pressure cone. I would suggest, however, that, after precautionary investigations (such as ventriculography) have proved negative, lumbar puncture should be carried out, because the impressions at ventriculography can be inaccurate in judging the intracranial pressure; in just the same way it is often difficult for the ophthalmologist to obtain an accurate impression of the intra-ocular pressure by palpation, the tonometric readings being frequently unexpected.

Recordings of the venous and arterial diastolic pressure at the optic disc could well be made part of the routine examination in patients with optic disc oedema or suspected to have raised intracranial pressure. This would allow direct comparison with the cerebrospinal fluid and systemic blood pressures. In an individual case it may not be of much value to take a single group of measurements which could give a misleading impression when phasic variations are taking place.

Summary

Cases of optic disc oedema associated with normal intracranial pressure, seen personally, are correlated with other reported cases. The suggestion is put forward that, by a simple alteration of the arterial and venous pressure ratios at the optic disc, papilloedema can be accurately simulated—lending support to the theories which suggest similar conditions in the aetiology of true papilloedema.

I should like to express my gratitude to my colleagues at the Midland Centre for Neurosurgery for their ready cooperation.

ADDENDUM

It was interesting to notice, on examination of the four cases shown this morning of drusen bodies in the optic nerve, that in three instances the venous pressure at the optic nerve head was raised while in the fourth it was normal: and in the three cases with raised venous pressure, the drusen were not especially notable on the disc head but appeared to be much deeper, whereas, in the case without raised venous pressure, the drusen were mostly superficially placed.

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NON-PIGMENTED INTRA-EPITHELIAL MELANOMA OF CORNEA*

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PRECANCEROUS melanosis of the surface epithelium is histologically analogous to Bowen's disease (intra-epithelial epithelioma) in that both are forms of carcinoma *in situ*, which sooner or later invade the subepithelial tissue, giving rise to typical malignant melanoma on the one hand and epidermoid carcinoma on the other. It has therefore been suggested by Ashton (1957a) that the term "intra-epithelial melanoma" would be more appropriate than "precancerous melanosis" and this name has been adopted throughout the present report.

Malignant melanoma, as a primary or secondary lesion, is exceedingly rare in the cornea. Von Hippel (1928) was able to collect only ten cases, and since that time seven more have been added to the literature (Castroviejo and Castroviejo, 1931; Reese, 1943; Rossi, 1951, 1952; Lister, 1951; Ashton, 1951; Davies and Bailey, 1954; François, Gildemyn, and Rabaey, 1956). Some of these malignant tumours apparently arose from pre-existing naevi, but in only one case was the lesion described as having derived from an intra-epithelial melanoma (François and others, 1956), although the histological description of the case of Castroviejo and Castroviejo (1931) suggests that this lesion may also have had a similar origin.

The case reported here showed an intra-epithelial melanoma of one half of the cornea, which merged imperceptibly with similar changes at the limbus. A malignant melanoma was present at the limbus on the opposite side, but this was quite distinct from the corneal lesion, being separated from it by healthy corneal epithelium. The case is unique in that the lesions were completely non-pigmented.

Case Report

A married woman, aged 64, was first seen in March, 1953, complaining of persistent discomfort for some months in the left eye, which before this time had been completely normal.

Examination.—The left eye was irritable with a number of superficial punctate infiltrations confined to the lateral half of the cornea. The right eye was normal.

* Received for publication April 4, 1957.

Diagnosis.—The condition was thought to be a superficial punctate keratitis, and various treatments were adopted during the next few months, all without benefit.

Progress.—By October, 1953, the corneal infiltrations had increased in size and number, a marginal pannus had developed corresponding to the affected area of the cornea, and a small protuberant mass was seen at one point on the limbus. There was no glandular enlargement.

Operation.—The possibility of a neoplasm was now considered for the first time. The exuberant tissue at the limbus was removed and submitted for pathological examination and reported as a malignant melanoma. In November, 1953, the left eye was excised, care being taken to remove a wide area of conjunctiva corresponding to the lesion.

Result.—Up to December, 1956, there had been no local recurrence and the patient remains in good health.

Histology

(*1*) *Limbal Biopsy.*—Section shows a nodule of tissue covered on one side by a thin layer of epithelium. The subepithelial tissues are packed with malignant melanotic cells arranged in whorls, sheets, and columns (Fig. 1).

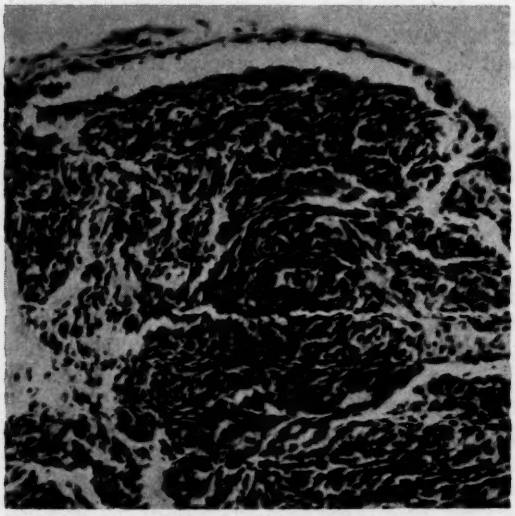

FIG. 1.—Limbal biopsy, showing subepithelial tissues densely infiltrated by malignant melanotic cells. Note the small clusters of malignant cells in the deeper layers of the covering epithelium. Haematoxylin and eosin. ×120.

There is no pigmentation, and the predominant cell, which is of the spindle type, is markedly pleomorphic (Fig. 2, opposite). A few clusters of cells lie within the deeper epithelial layers resembling those seen in intra-epithelial melanomata.

(*2*) *Whole Eye.*—Section of the eye shows a healed biopsy scar at the limbus on the temporal side with a few malignant spindle cells lying in the surrounding stroma. At the limbus on the opposite side and extending into the bulbar conjunctiva there is an extensive cellular proliferation within the deeper layers of the epithelium (Fig. 3, opposite).

There is considerable cellular pleomorphism, cytoplasmic vacuolation, and some pyknosis; many of the cells are grouped into small nests or clusters (Fig. 4, opposite).

Agglomerations of cells are bulging into the underlying limbal stroma, but as yet have not broken away from the main mass and the lesion is apparently still in the precancerous intra-epithelial phase.

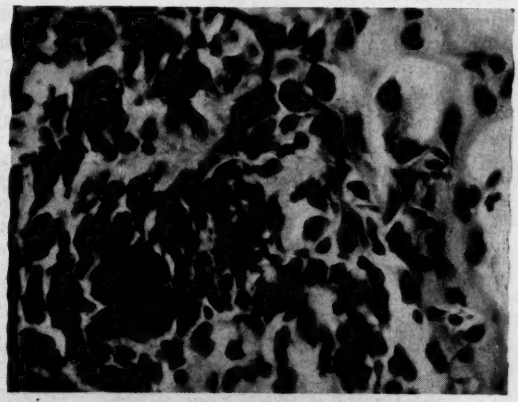

FIG. 2.—Limbal biopsy. High-power view of *Fig. 1*, showing typical clusters of non-pigmented malignant cells. Haematoxylin and eosin. ×390.

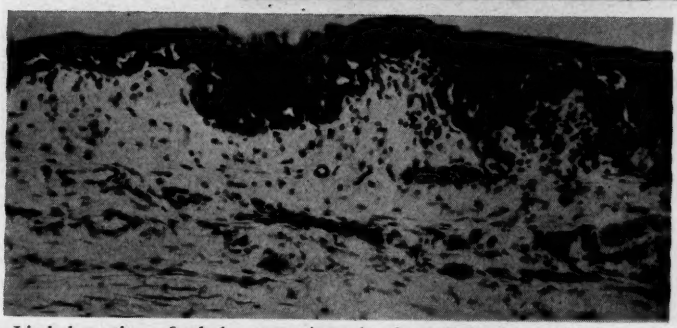

FIG. 3.—Limbal portion of whole eye-section, showing cellular intra-epithelial proliferation. The cornea is on the left. Haematoxylin and eosin. ×120.

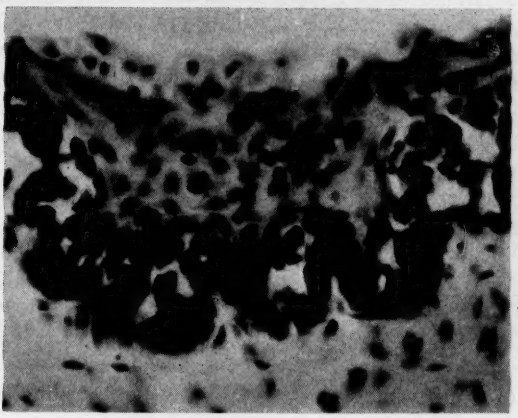

FIG. 4.—High-power view of a cluster of proliferating intra-epithelial cells seen in Fig. 3. Haematoxylin and eosin. ×360.

Within the basal layers of the corneal epithelium, on this same side, extending as far as the centre of the cornea and merging with the limbal lesion, there is a continuous collection of tumour cells separated from the substantia propria by an intact Bowman's membrane (Figs 5 and 6). The cells are rather more pleomorphic than those of the limbal precancerous lesion, having no clear cytoplasmic spaces or nest-like arrangements and thus suggesting early malignancy, although in the absence of subepithelial invasion it is difficult to be certain of this change. As in the limbal biopsy there is complete absence of pigmentation.

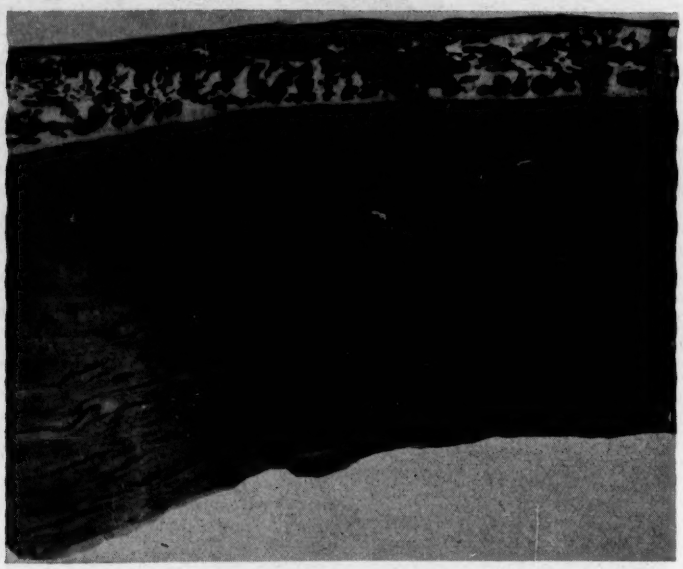

FIG. 5.—Tumour cell proliferation within deeper layers of corneal epithelium separated from substantia propria by an intact Bowman's membrane. Haematoxylin and eosin. ×120.

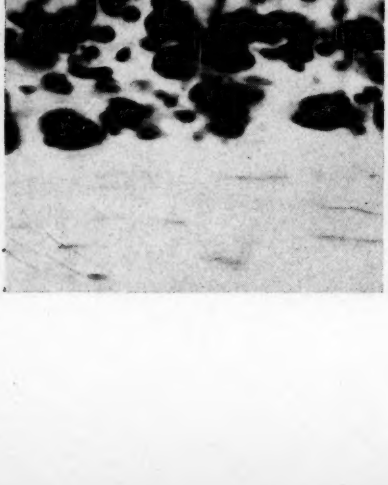

FIG. 6.—High-power view of Fig. 5, showing intra-epithelial cellular proliferation of the cornea. Haematoxylin and eosin. ×380.

Discussion

Malignant melanomata of the outer eye usually arise from either a pre-existing naevus or from an intraepithelial melanoma, and the distinction can frequently be made on the clinical history alone. A pre-existing naevus, for instance, will have been noted from childhood, whereas intra-epithelial melanoma is a disease of the fourth and fifth decades and arises in a previously healthy epithelium. Furthermore, a conjunctival intra-epithelial melanoma is characteristically diffuse, and, by the time malignant change has developed, may have spread to involve the whole of the bulbar and palpebral conjunctiva together with the caruncle and adjacent skin of the lids (Greer, 1954).

The histological picture of intra-epithelial melanoma is now well known and, except for the absence of pigmentation, our description is typical of the condition. The history and histological picture of this case leave no doubt that the whole neoplastic process began as an intra-epithelial melanoma of the conjunctiva. This condition, even if widespread, rarely extends onto the cornea, and it is surprising that this feature has not attracted more attention. The reason for this corneal immunity is unknown, although of course there are elsewhere in the body other examples of freedom from direct spread by a neoplasm. Blondis (1946) believes that at the limbus there is a physiological barrier which breaks down in disease to permit the migration of melanocytes into the cornea.

The possibility that some of the reported cases of primary melanomas of the cornea have in fact arisen from the limbus, cannot be completely excluded. Even in the well described case of Davies and Bailey (1954) doubt must be cast upon the corneal origin as the authors describe the basal cell proliferation extending to the region of the limbus. A more certain case of a corneal primary was that of Blanquinque (1892), who described a "melanosarcoma" in a woman aged 60. The patient had noted a red spot near the centre of the cornea 20 years earlier, and this remained quiescent for 11 years, gradually increased in size for 9 years, and at the time of excision had extended to 1 mm. beyond the limbus. Reese (1951) published a photograph of a corneal melanoma and stated that serial sections showed no limbal invasion.

The origin of true primary corneal epithelial melanomas, if they exist, is not apparent. Reese (1951) believes that they arise either from the Schwann cells of the corneal nerves or from the basal cells of the epithelium, thus embracing two opposing theories of the histogenesis of melanomata—a neural and an epidermal origin.

Recent work, however, has proved that melanogenesis takes place exclusively within the cytoplasm of the dendritic cells (Billingham, 1948, 1949; Billingham and Medawar, 1948, 1950, 1953), which pass under a variety of names, *i.e.* melanocytes, melanoblasts, and Langerhans' cells. They lie between and beneath the basal cells of the epidermis, and according to

Billingham (1948) have a neural origin. It follows, therefore, that if benign and malignant melanomata arise from these dendritic cells, as is now widely believed (Cappell, 1951; Lewis, 1954; Lloyd, 1954; Evans, 1956), they also must have a neural and not an epidermal origin.

Dendritic cells have not been found within the normal corneal epithelium, but Redslob (1922), using a silver stain and the "dopa" reaction, showed that "melanoblasts" are present in the epithelium of injured corneae. He was unable to obtain a specimen of a normal human cornea but found no evidence of "melanoblasts" in the normal corneae of rabbits, and this has recently been confirmed (Michaelson, 1952). Redslob (1922) found melanoblasts in one apparently normal cornea but on deeper section he was disappointed to find evidence of an old injury. He concluded that the presence of vascularization was necessary to bring melanoblasts into the cornea.

Ashton (1957b) is not convinced that this is the complete explanation. He suggests that the melanocytes may be carried into the cornea from the limbus (where they can be demonstrated in normal eyes) by the epithelial slide that follows corneal injury. As is well known, this sliding movement may be extensive, with migration of epithelial pigment and goblet cells for some considerable distance into the cornea (Duke-Elder, 1954). It is therefore likely that injury is the predisposing cause of corneal melanomata which appear to be primary, and since there was no evidence of trauma in this case the corneal lesion is considered to be an extension from the limbal intra-epithelial melanoma.

The other feature of interest is the complete absence of pigmentation. Lloyd (1954) states that Fontana's silver stain will demonstrate the presence of melanin granules in most non-pigmented melanomata; but on the tissues from the patient here reported the technique gave negative results. Amelanotic melanomata have been reported elsewhere in the body recently by Andrews (1950), Bhende (1952), and Hecht (1953). Malignant melanomata of the choroid are occasionally non-pigmented, but within the conjunctiva and cornea lack of pigmentation must be exceedingly rare.

This feature frequently makes the diagnosis of such lesions extremely difficult, both clinically and histologically, especially if the cellular pattern is unusual as occurred in the case of Hecht (1953). It was fortunate that the histological picture in our case was so typical that the absence of pigmentation caused no confusion.

This case was under the care of Mr. E. F. King, to whom I am greatly indebted for the clinical details.

I wish to thank Prof. Norman Ashton for his permission to publish the pathological report and for his encouragement and help in preparing the paper.

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BLOOD SUPPLY OF THE OPTIC NERVES AND CHIASMA AND ITS CLINICAL SIGNIFICANCE*

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PART I THE OPTIC NERVE

The blood supply of the optic nerve has been a subject of controversy for at least 200 years, since the description of the arterial circle around the optic nerve head by Zinn (1755) and Haller (1754). Numerous papers have been written concerning the subject and in the past few years new techniques of injection, micro-angiography, and dissection and Neoprene casts have resulted in further papers on the subject. Older writers, using relatively crude techniques, differed widely in their interpretation of the blood supply of the nerve. What is, perhaps, more surprising is that modern workers using highly elaborate and skilled techniques have also differed considerably in their descriptions of the vascular supply. The differences are especially noteworthy when considering the central vessels of the optic nerve, the anastomoses around the optic nerve head, and the blood supply of the intracranial portion of the optic nerve.

All observers are agreed that the optic nerve is surrounded by a pial plexus of vessels and also that it contains some system of central vessels. There is disagreement concerning the contribution made to these systems by individual vessels and the degree of anastomosis between them.

Most workers seem to be agreed that the outer layers of the optic nerve in its orbital portion are nourished from a capillary pial plexus. This is contributed to mainly by twigs from the ophthalmic artery, and its branches the long and short posterior ciliary arteries. François and Neetens (1954) postulate also twigs from the lacrimal artery. How much of the nerve is supplied by this pial plexus seems to be a matter of speculation. Steele and Blunt (1956) and many other writers have noted that branches from this plexus appear to penetrate the optic nerve for short distances only, so that it may be inferred that probably less than half the fibres are supplied from this source and that they are those situated peripherally in the nerve. There seems to be no convincing evidence from these studies that there is any horizontal or vertical division of blood supply in this peripheral portion. The only other point at issue over the pial plexus is the contribution from the central artery itself. All writers other than François and Neetens agree that this vessel

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gives off branches to the pial plexus before its entry into the optic nerve, so that it may be assumed that in most cases some local contribution, presumably to the inferior peripheral fibres, derives from the central artery outside the nerve.

The main controversy concerns the origin, distribution, and method of branching of the central vessels. Branches have been described from the central artery itself within the nerve (Steele and Blunt, 1956; Wybar, 1956). A central artery of the optic nerve itself has been described arising from the ophthalmic artery (François and Neetens, 1954), and many authors have described anterior and posterior axial vessels in the nerve derived from the central artery either before or after its entry into the optic nerve. Though many workers have denied the presence of any branches from the central retinal artery within the optic nerve, no one who has studied the excellent illustrations of Steele and Blunt (1956) and of Wybar (1956) can deny their existence. In what proportion of subjects these branches exist must remain a matter of speculation and is not of great importance.

Most workers have found axial vessels entering the optic nerve with the central retinal artery and arising from it just before its entry. In some cases the origin may be after the vessel has entered the nerve and in some cases its origin may be far back and may arise from the ophthalmic artery itself rather than from the central retinal artery (François and Neetens, 1954). The distribution of the axial vessels, variously termed the collateral arteries of the central retinal artery, anterior and posterior axial arteries, or central artery of the optic nerve, has also been the subject of controversy. Earlier writers tended to adhere to the description of the axial vessels running forward and backward throughout most of the length of the orbital optic nerve, and such a vessel was described by Vossius (1883). Since then varying descriptions have been given, and it seems commoner to find a long branch directed posteriorly as far as the optic canal than a long anterior branch (Wolff, 1948). Others, notably Wybar (1956), whilst demonstrating the presence of axial branches, deem them to be only short vessels with a limited area of supply.

To collate information on this point, therefore, it seems probable that the central core of the optic nerve within the orbit is supplied in a variety of ways. Contributions may come from the intraneural part of the central artery of the retina, from axial vessels derived from the central artery before its entry into the optic nerve which may send out anterior and posterior axial branches, and sometimes from a separate central artery of the optic nerve arising from the ophthalmic artery in the orbit. All such systems have been described by highly skilled and competent observers and must be accepted as present in the specimens they dissected. The variability in supply must merely reflect the vagaries of human anatomy, and the only fault lies in our own scientific or Procrustean attempts to cover in a single rigid description a system of considerable variation.

The same situation appears at the optic nerve head. Here the pial plexus receives contributions from the circle of Zinn, and the point at issue is to what degree, if any, arterial anastomosis takes place between the choroidal and optic nerve arterial supply, for all are agreed that the capillary anastomosis seems to be present. The answer can only be that such anastomosis is present in some cases but probably not in most.

The blood supply of the intracranial optic nerve has also been subject to considerable difference of opinion. Dawson (1948) described and illustrated a sizeable branch of the ophthalmic artery running along the infero-medial aspect of this portion of the nerve and giving off branches to it. Steele and Blunt (1956) have denied this and state that, in 34 dissections in which this vessel was especially searched for, it was found in none. On the other hand, they assert that the major supply to this area of the nerve is from a branch of the anterior superior hypophyseal artery running along the under side of the nerve. They did find small branches from the ophthalmic artery going to the nerve, but these were very small and only found in the region of the optic canal. Other writers have noted contributions from the anterior cerebral, anterior communicating, and internal carotid arteries. We must be faced here with a similar situation and can only conclude that all the vessels named do, on occasion, contribute to the blood supply of this portion of the optic nerve, but that the individual variations are much greater than one commonly supposes. My own observations during operations on this region add yet another possibility. I have frequently noted a considerable contribution to the upper surface of the optic nerve from the ophthalmic artery and from the anterior cerebral artery. Both these vessels are relatively large, and easily seen by the naked eye during surgical operations, and have been observed a great many times. Viewing a vessel on the surface of the nerve does not necessarily imply that it supplies this area of the nerve but such would seem probable.

Of the arteriolar pattern relatively little has been said. Most of the supplying vessels are small and on entry into the nerve break up into small arterioles. Perhaps little more than this can usefully be said. Capillary patterns have been studied with great care by François, Neetens, and Collette (1955), and François and Neetens (1956). They have described, in the orbital portion of the optic nerve, a system of capillaries surrounding fibre bundles and appearing in transverse sections as pentagonal boxes surrounding these bundles, whilst in longitudinal sections these circles are connected by longitudinally running capillaries and thus the appearance is one of long rectangles. In the intracranial portion of the optic nerve, this regular pattern is soon lost and gives way to the more irregular meshworks of the chiasma.

Certain inferences can be drawn from this pattern of vascularization which may have clinical significance. Firstly, there seems to be a clear distinction in the orbital portion of the optic nerve between peripheral and central

supply. The peripheral fibres are supplied from a rich pial plexus to which many vessels may contribute. The central area, by contrast, appears to be supplied by fewer vessels and perhaps on occasion by one only, the central artery of the optic nerve or the collateral artery of the optic nerve. Disturbance of function from a vascular cause may well show, therefore, predominantly peripheral or central field loss. Again, one might imagine that the central area could be more vulnerable than the peripheral because a single small lesion, compressing, clotting, or otherwise interfering with blood flow in a single small vessel, could inhibit or destroy function in the whole of the central area of this nerve segment. Secondly, apart from this central-peripheral separation, there is no evidence in the arterial, arteriolar, capillary, or venous patterns to suggest segmentation of another kind. Thirdly, capillary patterns at least suggest that they are based on bundles of fibres and therefore the effects of ischaemia should be recognizable in field loss of a "nerve fibre bundle" type. Fourthly, in the intracranial portion of the optic nerve, the central-peripheral segregation and the fibre bundle capillary box disappear, so that one would expect these features of optic nerve lesions to disappear also. On the other hand, the blood supply in this region appears to be, in some cases at least, much more dependent on single vessels. Steele and Blunt found in their dissections that the anterior superior hypophyseal artery was so often the sole vessel of supply that it might conceivably be called the vessel of the intracranial optic nerve. Where this is not so it may well be replaced by another single vessel, the pre-chiasmal artery of Dawson. In these circumstances one might infer that this portion of the optic nerve was especially vulnerable to pressure from below and medially, or to any arterial disease that might affect either of these vessels.

The clinical aspects of the subject may now be discussed and the effects on optic nerve function of vascular disease, pressure, trauma, and so forth may be examined to see if they accord with the principles inferred from anatomy.

Practitioners of clinical perimetry are accustomed to recognize at least two stigmata of vascular disease in the optic nerve, fibre bundle defects and horizontal hemianopias, usually in the lower field. It may be as well to consider a group of vascular lesions to determine how justified this assumption may be.

The hemianopic theory probably originated from those peculiar cases in which vision is lost after massive haemorrhage, often gastric in origin. When vision and field are recovered or preserved, the field pattern is characteristically that of an inferior, or less commonly superior, hemianopia. The line of demarcation lies strictly along the horizontal meridian and it is usual to find in addition generalized depression in the preserved field. No one seems to have determined whether this lesion lies in the optic nerve or the retina, certainly the horizontal line of demarcation would suggest a retinal origin. In some cases, however, the picture is not so clear cut. In one example the fundus was observed throughout the episode and the retinal

vessels were noted to be normal. In this case the line of demarcation did not follow exactly the horizontal meridian and it seems probable that the lesion lay in the optic nerve. Another and clearer example is the following:

A young man suffered from angiomatosis of the optic nerves, some portion of the angioma being visible on the optic nerve head. He was subject to periodic attacks of inferior hemianopia in either eye lasting about half an hour. On one occasion the hemianopia in one eye failed to recover as usual and left a permanent inferior hemianopia with clear-cut margin along the horizontal meridian. The retinal vascular tree during this time was noted to be quite normal.

A not dissimilar picture may be seen in the so-called arterio-sclerotic atrophy of the optic nerve, though in this instance the evolution may be a gradual one. A similar example has been seen in association with arterial hypertension; in this instance the onset of the hemianopia was sudden and had all the characteristics of an arterial thrombosis (*Fig. 1*).

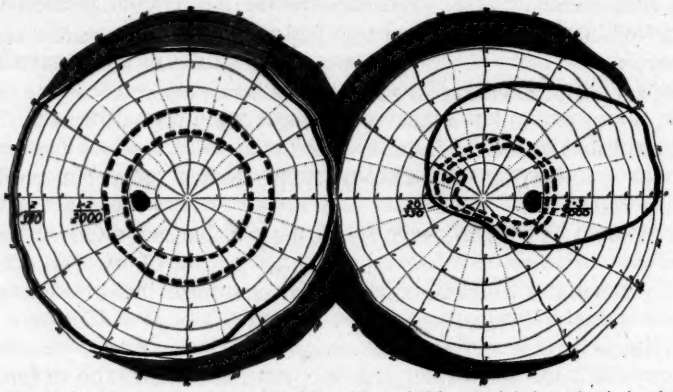

FIG. 1.—Visual field in a case of hypertension with probable arterial thrombosis in right optic nerve. Sudden onset of field loss without detectable change in retinal vessels.

These are but a few examples from a vast store of material in the literature and in personal records. Such clinical experience suggests rather strongly that there is some segmental arterial supply in the optic nerves whereby the blood supply of one horizontal half of an optic nerve may be cut off by a single lesion. In many of such cases the central area of the field is spared, but in some this is also affected. None of the anatomical work reported has provided any explanation of this clinical experience.

Another fact which has emerged clearly from anatomical work is that the central and peripheral areas of the optic nerve have a separate blood supply, though variations are considerable and there may be some anastomosis. In such conditions one might expect to find separate examples of both central and peripheral field loss in vascular lesions, and, in my experience, such is the case. The commonest type of field loss in so-called arterio-sclerotic optic atrophy is the appearance of multiple indentations in the peripheral field.

These most commonly affect the lower field but are also seen in the upper field (Fig. 2).

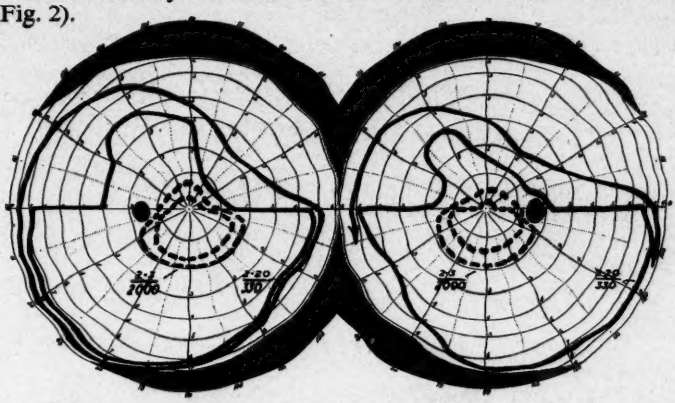

FIG. 2.—Visual fields in a case of arterial thromboses in both optic nerves associated with generalized arteriosclerosis. Gradual progression over 18 months.

Usually both eyes are affected, but occasionally the condition may appear in one eye some time before the other is affected. Many of these cases have peripheral field defects connected with the blind spot and suggestive of nerve fibre bundle defects. Conversely, isolated defects in the central field, or fibre bundle type, may be seen. These are difficult to distinguish from the fields of chronic glaucoma (Hughes, 1954; Fig. 47).

Additional evidence may be adduced from the indirect injuries of the optic nerve. The general consensus of opinion now favours a lesion in the optic canal whereby the nerve is displaced or twisted and small arterial twigs going to the pial plexus are torn. Inferior hemianopia is the commonest field defect and usually the macular area is spared as one would expect from the anatomy of this area. Fibre bundle defects and generalized depression are seen frequently and superior hemianopia occasionally (Hughes, 1945).

A great many lesions of the optic nerve produce field loss which has neither the features of horizontal hemianopia nor those of fibre bundle defects. Compression of the nerve is a particular example and has puzzled many generations of clinicians. Most writers are agreed that some form of central field loss is the commonest feature of compression whether the lesion be intracranial or orbital. I have found that a central scotoma with quadrantic features and often extension to the blind spot is so common as to be almost diagnostic of compression of the orbital portion of the nerve though less frequent with intracranial lesions (Hughes, 1954).

In most situations where compression takes place, the fibres subserving the central area of the field are placed in the centre of the nerve and the reason why they should be affected whilst peripheral fibres adjacent to the tumour are involved relatively late is a riddle as yet unsolved. The position with regard to the various theories has been admirably stated by Mooney and

McConnell (1949). Meadows (1949) has suggested that such cases may be seen only in a late stage when break-through to the periphery has occurred and that even in the early case some peripheral loss may be present. This does not accord with my own experience and even when careful quantitative perimetry is done in the early case no peripheral defect can be detected though the central field changes themselves may be slight (Fig. 3).

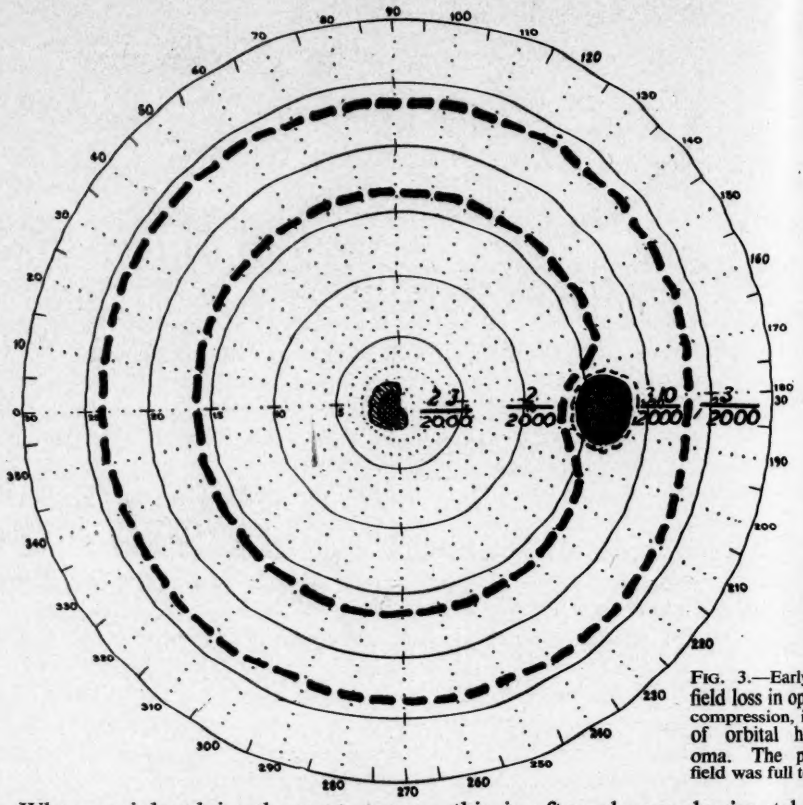

FIG. 3.—Early cen
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When peripheral involvement appears this is often along a horizontal meridian and not always related to the maximum area of the central scotoma, *i.e.* it is not a "break-through" (Fig. 4, opposite).

In very late stages of compression the whole of the central and intermediate field disappears, leaving only a rim of dim vision around the periphery, and this rim does not usually have hemianopic characteristics (Hughes, 1954; Fig. 95). It is difficult to explain these changes. Many writers have made a vague suggestion that "vascular factors" must play a part. Mooney and McConnell (1949) have stressed that vascular changes in the region of the optic canal produce predominantly peripheral field defects and

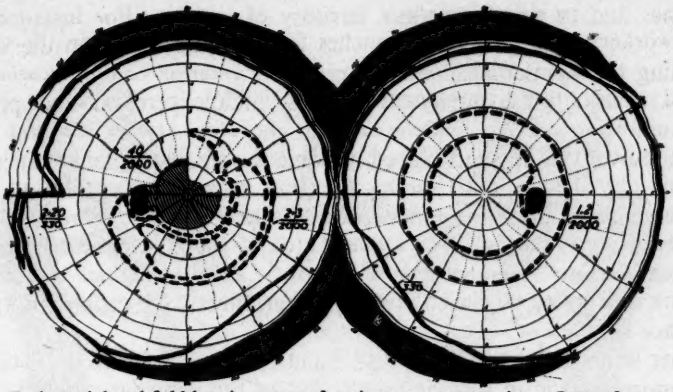

FIG. 4.—Early peripheral field loss in a case of optic nerve compression. Loss of central field is already far advanced before peripheral loss occurs and this is not related to break-through of a scotoma. From a case of "pseudo-tumour" of the orbit.

instance optic nerve injuries as an example of this. They have also made an interesting contribution in suggesting that central field loss is often associated with compressing lesions near the optic canal intracranially which may displace the nerve in relation to the canal and compress, secondarily, the ophthalmic artery. This hypothesis is amply supported in my own experience and may provide an explanation for the intracranial lesions at least. Definitive lesions of the ophthalmic artery producing central field loss have not, however, been reported and it is difficult to see why compression of an artery supplying all parts of the nerve should have this effect. The position with regard to intra-orbital lesions is more difficult. Here the common site for tumours is above the nerve, and the arterial supply to the central area lying beneath the nerve is cushioned from the tumour by the nerve itself and protected from counterpressure by soft tissues beneath. It is possible that downward and lateral pressure might nip the ophthalmic artery against the anterior edge of the optic canal and yet the type of field loss seems to be so constant wherever the tumour may be.

These field defects betray neither of the features associated with vascular lesions, horizontal hemianopias, and fibre bundle defects, and the bulk of evidence would suggest that the field loss is not of vascular origin but must be related to compression or kinking of the nerve fibres themselves.

PART II. THE CHIASMA

In the chiasma there is much greater agreement on the pattern of vascularization, at least as far as the central area is concerned; some disagreement still remains concerning the anterior chiasmal angle and intracranial portion of the optic nerves.

Earlier writers recognized a vascular network running upwards around the pituitary stalk from which many small branches ran to the chiasma itself. More recently writers have been concerned to map out individual supply

branches and to determine their territory of supply. For instance, most early workers have described branches from all the vessels in the vicinity, including the ophthalmic, anterior cerebral, anterior communicating, and carotid artery, the hypophyseal arteries, middle cerebral and posterior communicating arteries, and the anterior choroidal artery. Several writers have admitted that the part played by some is a small one, and François and Neetens (1956) state that, during injection experiments, exclusion of the middle cerebral, posterior communicating, and anterior choroidal arteries resulted in non-filling of only a small area of the chiasma in the postero-temporal region. Nevertheless, they adhere to the original idea that the chiasma is supplied by eleven arteries, either directly or secondarily through a surface network of vessels.

Other writers, notably Steele and Blunt (1956) and Dawson (1948), have been more specific in their interpretation of this vascular network. They have described separate branches from the anterior cerebral and anterior communicating, from the carotid itself, and from the posterior communicating; in particular they cite the anterior superior hypophyseal artery from the carotid as being a major source of supply to the inferior surface of the chiasma.

One may, perhaps, justly combine the views so propounded in the following manner: On the inferior surface of the chiasma a major artery of supply is the anterior superior hypophyseal artery. This arises from the carotid, passes medially, and sends a major branch to the chiasma either directly or *via* the peri-infundibular plexus. It appears to supply much of the central region of the chiasma inferiorly and especially posteriorly and is claimed by Steele and Blunt to send forward a definitive branch to the under surface of the intracranial portion of the optic nerve which serves as its main supply. Branches also arise from the posterior communicating to supply (or at least enter) the postero-inferior angle of the chiasma (Fig. 5, opposite).

A separate branch arises from the medial surface of the internal carotid itself and enters the infero-lateral aspect of the chiasma. This is an important branch, for it is the only single artery of supply and it enters a region of the chiasma in which are situated the fibres from the uncrossed upper nasal field.

Most writers seem agreed that the superior aspect of the chiasma and its anterior angle are supplied by branches from the anterior cerebral and anterior communicating arteries. These arise from the anterior aspects of the vessels concerned and pass forward to enter the upper surface of the chiasma, some passing around the anterior margin. There is, however, some divergence of opinion concerning the vessels in this region. Dawson (1948) has described an anterior chiasmal plexus or arcade. This he states is composed of branches from the anterior cerebral and anterior communicating arteries, and is also largely contributed to by a vessel from the ophthalmic artery.

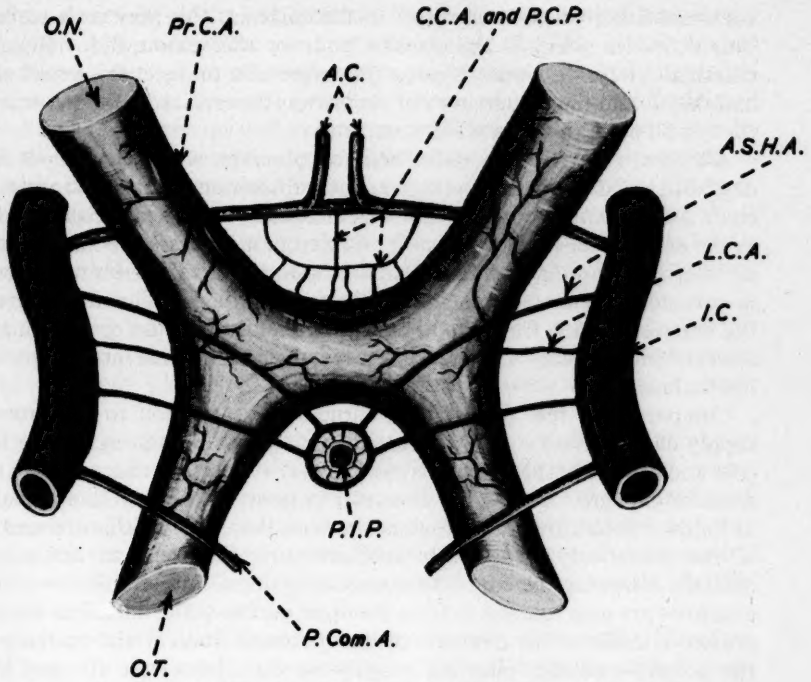

FIG. 5.—Schematic representation of the blood supply of the inferior surface of the chiasma. On the right side of the diagram the optic nerve is supplied by a twig from the anterior superior hypophyseal artery, on the left side by twigs from the ophthalmic artery (the prechiasmal artery of Dawson).

O.N.	Optic nerve	P.I.P.	Peri-infundibular plexus
O.T.	Optic tract	A.S.H.A.	Anterior superior hypophyseal artery
I.C.	Internal carotid artery	L.C.A.	Lateral chiasmal artery
A.C.	Anterior cerebral artery	C.C.A.	Central chiasmal artery
P.Com.A.	Posterior communicating artery	P.C.P.	Prechiasmal plexus

This he describes as arising from the intracranial portion of this artery, running along the infero-medial aspect of the optic nerve and the antero-inferior margin of the chiasma to anastomose with its fellow of the opposite side and with the anterior cerebral branches. He names this the pre-chiasmal artery. Others, however, have failed to find this vessel in dissections. In fact, most writers state that the intracranial contribution of the ophthalmic artery is insignificant and perhaps confined to one or two small branches to the optic nerve near the canal. François and Neetens (1956) agree with the presence of an anterior chiasmal arcade, but have found it to be supplied by branches of the carotid, the anterior cerebral, and the anterior communicating; in addition branches of all these vessels might go direct to the chiasma. They found, in a third of their dissections, a specific artery from the anterior

communicating which went direct to the chiasma; this they state to be the only definitive artery to the chiasma and, for this reason, they name it the chiasmal artery. On one occasion they were able to inject this vessel alone, and they found that its territory of supply was the central area of the chiasma, though filling in this area was incomplete.

All the workers cited are skilled and able observers who have without doubt described accurately the findings in their dissections. One can only conclude that the morphology of chiasmal arteries is very variable, at least as far as the anterior region is concerned, and that in most cases it is supplied from an arterial plexus but that in occasional cases definitive arteries may be seen: in particular the lateral branch from the carotid (the lateral chiasmal artery), the anterior branch from the anterior communicating (the central chiasmal artery), and possibly in rarer instances the prechiasmal artery from the ophthalmic.

Comparatively few writers have turned their attention to the arteriolar supply of the chiasma or to the capillary pattern, and amongst these François and Neetens (1956) and Dawson (1948) stand out. Surprisingly there seems to be agreement on the arteriolar pattern. This may be summarized as follows: Inferiorly branches enter the central area of the chiasma and tend to run posteriorly. Superiorly and anteriorly they tend to run postero-medially following the course of the crossing fibres. In addition two sets of arterioles are seen running in from the optic nerves. The lateral set run back postero-laterally in the direction of the uncrossed fibres whilst medially they run postero-medially following roughly the direction of the crossing fibres. Posteriorly arterioles enter from the region of the optic tract and run to the lateral area of the posterior chiasmal angle.

One may assume that the lateral fanning branches are derived from the lateral chiasmal artery, whilst the central and anterior crossing arterioles are derived from the anterior chiasmal area and central chiasmal artery. The inferior arterioles are presumably derived from the anterior superior hypophyseal artery. It is surprising that some arterioles are not seen running anteriorly towards the optic nerve if Steele and Blunt are correct in their descriptions.

It would be difficult to predict from this description what might be the result of a specific vascular occlusion, the arterial supply is in large part plexiform, and loss of a single branch might be expected to have little effect on the whole. Certain features of the anatomy are, however, suggestive. In the first place, the supply to the inferior uncrossed fibres seems to be by a definitive artery situated infero-laterally and well out of reach of the usual compressing tumour from the pituitary fossa. This may well explain the preservation of the upper nasal quadrant in so many advanced cases. Secondly, the supply to the post-inferior border, the site of the macular crossing, seems to be supplied from below, possibly by the superior hypophyseal artery or a branch from the posterior communicating. Lesions in

this area, therefore, might be expected to produce early central hemianopic scotomata. If we accept the presence of a central chiasmal artery in some cases, then a small lesion anteriorly might well produce marked bitemporal loss without gross evidence of pressure. Again the occasional finding of marked bitemporal loss (as well as optic nerve defects on one side) in laterally placed lesions under the optic nerve might be explained by the presence, in that instance, of an ophthalmic branch, the pre-chiasmal artery of Dawson. Generally speaking, however, it will be seen that the arterial supply is closely related to the fibres it supplies, so that in a given instance it is hardly conceivable that one could separate the effects of direct pressure on fibres from those of occlusion of small arterioles.

The capillary supply seems to have relatively little importance. When viewing the chiasma in low magnification stained for vessels, one is struck by the apparent similarity between the fibre patterns and the vascular pattern.

Lateral capillaries run roughly antero-posteriorly as do the uncrossed fibres, whilst central capillaries, especially those anteriorly placed, sweep across the midline in just the way of the crossing fibres. The excellent work of François and his colleagues has shown that this is hardly the case, and they have divided the chiasma into no less than six paired zones, and one central zone, in which the capillary formation, including size of vessels, density, and method of branching, are all distinct.

One's first view of the chiasmal capillary network is probably, therefore, misleading. In particular, it is noteworthy that, though the central capillaries sweep across in a superficial resemblance to the crossing fibres, they do not intermingle or criss-cross in the centre as do the fibres. In addition, there is no evidence in the capillary network of the fibre loops in the optic nerve and tract so clearly described by writers on fibre anatomy.

Study of vascular pattern in the supero-inferior direction is also disapointing, for one so often sees in visual field loss a quadrantic element to a defect suggesting that there may be a vascular raphe corresponding to the horizontal meridian in the chiasma as well as the retina and optic nerve. No evidence of such a raphe has been found by me or by other writers on the subject.

Comparatively little attention has been paid to the venous picture in writings on the vascularization of the nervous system, though, as most of us well know, the effects of venous thrombosis or obstruction may be as lethal to neural function as arterial obstruction. According to Dawson (1948), venous plexuses are present above and below. Superiorly, the veins run upwards from the plexus to join the anterior cerebral veins running with the anterior cerebral arteries. Inferiorly, the veins run posteriorly and join a pre-infundibular vein or venous plexus from which a large vein runs postero-laterally on either side to join, eventually, the basal vein of Rosenthal. Really very little seems to be known of the territory of drainage of these vessels and further study might well repay the time spent on a difficult technique.

It might be useful now to approach the subject from another angle and examine the clinical experience of visual field loss to ascertain whether there are any specific features which can be ascribed to a vascular disturbance.

In the majority of instances field loss is associated with gross disturbance of anatomy by tumour or other process and the usual reaction is one of surprise that the disturbance of function is not much greater than it is. As has been suggested above, arteries and veins are mostly closely related to the fibres they supply and it is impossible in a given instance to separate the effects of direct compression, arterial and venous obstruction. There is considerable difference of opinion on the role of vascular obstruction, for François and Neetens (1956) have stated that in their view field loss is nearly always due to the mechanical effects of compression rather than to vascular obstruction, whereas Harrington (1956) believes that vascular obstruction is the major cause of field loss in cases of brain tumour. It may, therefore, prove fruitful to examine certain categories of field loss to see what light they may throw on the vascularization of the optic pathways or, in other circumstances, whether a knowledge of the anatomy of vascular supply can explain apparently incongruous clinical findings. Obviously it will be worthwhile to examine field defects presumed to be due to disease of the vessels themselves either from thrombosis, spasm, or the effects of trauma. Some cases in which the field loss appears to implicate fibre tracts at a distance from the causative lesion may well repay study also, and, lastly, those cases in which the pattern of field loss, or more often preservation, are difficult to explain on purely mechanical grounds.

Instances of direct involvement of chiasmal vessels by arterial disease are rare in the literature. The clinical interpretation in such cases is confused by many factors, not the least of which may be individual convictions. The role of arterial obliteration, primary neural degeneration, arachnoiditis, and compression from calcified plaques in vessel walls all have to be sorted out, and in the majority of cases to ascribe a given field defect to one particular factor proves to be impossible, even with the aid of histological examination. In the few cases I have seen and have felt reasonably satisfied that arterial obliteration was the primary factor the field loss has been a simple bitemporal depression affecting the upper quadrants principally (Fig. 6, opposite).

In this case the subject was an elderly man with general arteriosclerosis, the mode of onset extremely sudden and followed by a period of improvement over some weeks. In fact, the clinical features, general condition of the patient, and subsequent history were all suggestive of a small arterial thrombosis. I have since seen several such cases and, whilst the pathological confirmation is lacking in them all, this clinical experience suggests that there is some definitive arterial supply to the antero-inferior region of the chiasma. The work described above would tend to support this view and the anatomical and clinical material taken together support the hypothesis that there may be, at least in some cases, a definitive artery of supply to this area of the chiasma.

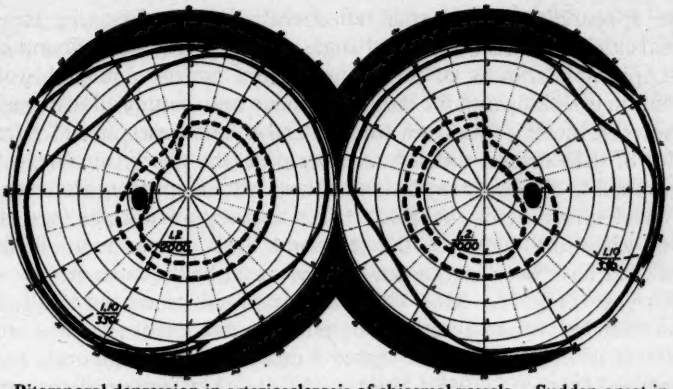

FIG. 6.—Bitemporal depression in arteriosclerosis of chiasmal vessels. Sudden onset in a man of 74 with some improvement during the next 6 weeks.

Whether this is the chiasmal artery of François and others derived from the anterior communicating, or the pre-chiasmal artery of Dawson derived from the ophthalmic, there would seem to be some divergence of opinion. Other views have been expressed, however, as to the characteristic visual field loss in arteriopathic lesions, and Traquair (1948) states that arterial obstruction and small softenings in the chiasma may be responsible for bitemporal hemianopic central scotomata with normal peripheral fields. This work suggests that other areas of the chiasma may have a definitive arterial supply. Each individual has only a small experience of such cases and it seems likely that only the pooling of experience and especially of pathological material from many sources is likely to advance our knowledge on a sound basis.

The mechanism of indirect chiasmal injury remains somewhat of a mystery. In reported cases operative inspection has not demonstrated any antero-posterior tearing such as one might expect from the clinical picture (Hughes, 1945), though such, indeed, has been described (Rand, 1936). Traquair, Dott, and Russell (1935) were the first to suggest that chiasmal dysfunction might be accounted for by damage to small arteries and subsequent thrombosis. In most cases the field loss is bitemporal and complete, though where some visual field is spared it is usually in the lower quadrant and might compare with the arteriopathic cases described above. In some personal cases there has been sparing of central temporal vision and this would suggest that damage has been confined to the territory of the chiasmal or pre-chiasmal artery, but perhaps this is special pleading and as far as I know there is no histological evidence available on this point.

In a case previously reported (Hughes, 1943), there was apparent injury to the anterior chiasmal angle. In the affected eye only an upper nasal field remained; in the opposite eye the upper temporal quadrant was depressed. I have since seen several such cases and in all but one the optic nerves and

chiasma appeared to be normal at operative inspection. In the single abnormal case the anterior chiasmal angle appeared grey and soft and had the macroscopic appearances of infarction. Here, unfortunately, histological confirmation is lacking and the lesion may have been simply a local contusion.

Pressure on the chiasma from tumour and other causes provides frequent examples of field defects difficult to explain on the purely mechanical basis of compression. Noteworthy amongst these is the apparent immunity of the upper nasal quadrant of the field. Even in very advanced cases of compression, when the whole of the temporal fields, lower nasal fields, and macular area have been lost, a small upper nasal field quadrant may remain intact for years (Fig. 7). Most chiasmal compressions are the result of compression from below and anteriorly by pituitary adenomata and the sequence of field loss is well recognized: upper temporal, lower temporal, and then lower nasal (Traquair, 1948; Hughes, 1954). Yet survey of the anatomy would suggest in theory that spread should be expected from the temporal field to the upper nasal, for the lower lateral fibres are those most closely associated with the tumour, and the tendency for the optic nerves to be rotated laterally would bring these fibres into even closer contact with it. Many have commented on this phenomenon and most are agreed with the sequence of events, though Traquair (1948) states that occasionally the upper nasal quadrant may be affected at a relatively early stage and quotes a case of Hirsch in which there was retention to a late stage of small symmetrical fields in the lower nasal quadrant.

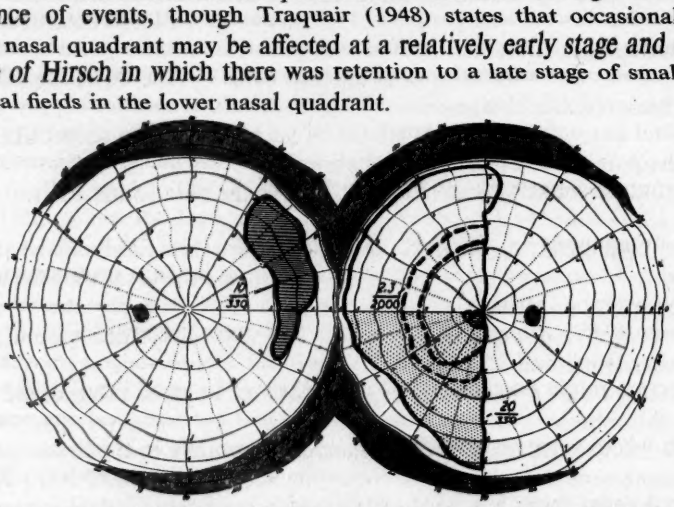

FIG. 7.—Residual upper nasal island of vision on the left in a case of chromophobe adenoma of the pituitary.

Many explanations of this field defect have been put forward, but none is wholly satisfactory. That the nasal field *per se* should be affected late is not difficult to understand. The lateral fibres are far removed from tumour pressure and only held against the tumour by the checking action of crossing medial fibres. As soon as these have been destroyed the lateral chiasmal tracts can easily be separated and can accommodate themselves to the growing tumour. Such a sequence of events is often reflected in an apparent

halt in the progress of a field defect after the temporal fields have been lost. Why the upper nasal field should be affected after the lower and often remains invulnerable for so long a time is much more difficult to explain. Traquair (1948) suggests that the upper fibres lie more medially in the lateral region of the chiasma and may so come to be affected before the lower fibres. Even if this explains the sequence of field loss, it offers no reasonable solution of the problem of the invulnerable upper nasal quadrant.

Other writers, notably Rucker and Kernohan (1954), have implicated the anterior cerebral arteries and have suggested that counter pressure of these vessels on the upper surface of the optic nerves or chiasma, often with deep grooving, may explain the lower nasal field loss. Much interest has been taken recently in this concept though the condition was described over a hundred years ago (Turck, 1852). Operative inspection of a large number of pituitary tumours has led me to believe that this factor plays rather a small part in the development of field loss. In many of the cases reported the lower nasal field defect has occurred early and is present even when quite large areas of the temporal field are retained. As an explanation of combined lower nasal and temporal field loss in an early stage of pituitary tumour compression, I feel it is quite adequate, but I doubt whether it is an explanation of the usual orderly sequence of field loss in these tumours. In one example a deep groove was present on the optic nerve but there was no associated field loss (Fig. 8).

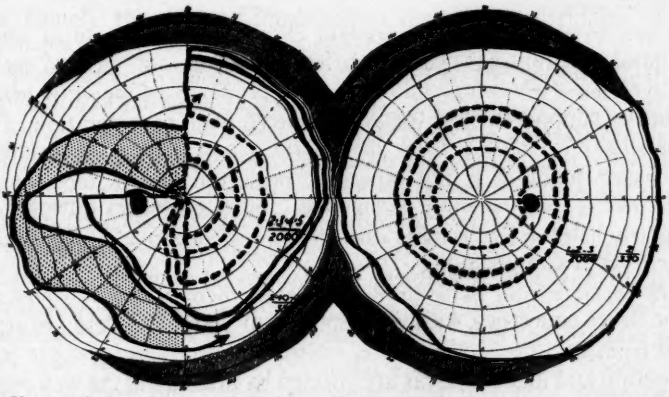

FIG. 8.—Chromophobe adenoma of pituitary affecting chiefly the left optic nerve. Operation disclosed a deep groove on the right side of the chiasma, but the visual field in the right eye had been normal only 12 hours before operation.

Other writers have suggested that the phenomenon is due to displacement of the optic nerves against the firm walls of the optic canal. This may well explain some cases of nasal field loss and, if the nerves were to be rotated laterally as suggested, then pressure from this cause might well fall on the upper lateral fibres. I would still doubt that this is the "normal" course of events, though it may well, like the previous example, explain the early

lower nasal field loss seen in some cases. Operative inspection of the nerves and chiasma in these cases has revealed a fairly common phenomenon. The crossing fibres in the chiasma are grey, fragmented, and hidden behind the mass of the tumour, the medial aspects of the optic nerves have the same, grey, avascular appearance. At the junction of the chiasma and lateral uncrossed fibres, there is a triangular bluish-grey area. This appearance is given by thinning of fibres in this region and in some instances tumour may be seen protruding through this area. The lateral bundles, presumably comprising the uncrossed fibres, appear normal, pink, and well-vascularized. This presumably, is the early stage of separation of crossed from uncrossed fibres by the tumour. Yet the explanation cannot be entirely mechanical for much of the grey area does not appear to be directly compressed by the tumour. The appearances may well be produced by infarction of this area leaving the lateral uncrossed fibres well-vascularized. Turning now to the anatomy of chiasmal vascularization, an important contribution may well be that of Dawson (1948) who, after many careful dissections, came to the conclusion that a branch from the carotid artery to the lateral and inferior aspect of the chiasma was the only definitive chiasmal artery he could identify. In just such a place we would expect to find the inferior uncrossed fibres from the upper nasal field. The fact that these fibres may have a definitive blood supply coming from a lateral aspect where it would be least subject to pressure from a midline tumour may well be the usual explanation of the preserved upper nasal quadrant.

In a few cases one sees an apparent chiasmal compression in which the upper nasal quadrant has been lost before the lower. In most of such cases in my experience the compression has been primarily of the optic nerve. A little further support for this thesis is obtained from consideration of lesions compressing the chiasma from the lateral aspect. In such instances the upper nasal quadrant is not especially preserved and in fact some cases suggest that it may be more vulnerable. Such evidence, however, is not particularly helpful, for in these cases the fibres concerned are subject to the direct effects of compression.

From consideration of compression fields, therefore, one would suspect that the fibres concerned with the upper nasal peripheral field are especially exempt from the effects of pressure, whilst the lower nasal and the whole of the temporal and macular areas are affected by pressure in the way one would expect. One can only conclude by suggesting that these fibres have some individual source of blood supply unaffected by the tumour.

It is common to find sharp quadrantic features in chiasmal field loss. It is very frequent in the macular field and often initial lower nasal loss in this region is of this character, the line of demarcation running exactly along the horizontal meridian (Fig. 9, opposite). This quadrantic loss is less often seen in the peripheral field, but none the less does occur and often, when greying is present, the line of demarcation runs exactly along the horizontal meridian.

Study of the fibre arrangement in the chiasma shows no evidence at all of

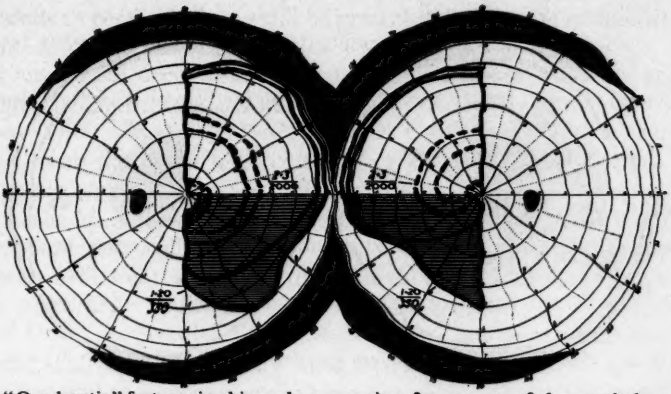

FIG. 9.—"Quadrantic" features in chiasmal compression, from a case of chromophobe adenoma of the pituitary.

any such quadrantic pattern and it is inconceivable, looking at sections of the chiasma, that this type of field loss could be due simply to the mechanical effects of pressure on such an inextricable tangle of fibres. Such quadrantic features in field loss are common in lesions of the optic nerve, especially with regard to the horizontal meridian, and are usually ascribed to "vascular" lesions. It may be that in such cases this is the effect of optic nerve compression, but in many instances the field loss has such clear chiasmal characteristics, namely, the clear-cut limitation by the vertical meridian. There is no evidence available so far as to supero-inferior fields of blood supply and, in fact, François and Neetens (1956) could find no characteristic features of vascularization in the supero-inferior plane. Be this as it may, the existence of two separate sources of supply, superior from the anterior cerebral and anterior communicating and inferior from the carotid, hypophyseal, and posterior communicating, may have some significance. One must, however, consider this problem as so far largely unsolved.

Compression of the chiasma produces scotomata of various kinds in the visual fields. They may be central and quadrantic, ring-like, circular, or peripheral, and may occur in any part of the field, though the central area is most common; scotomata extending into the nasal field are very rare and are probably due to super-added optic nerve compression. Those in the temporal field have one feature in common in that they never pass the vertical meridian, and this strongly suggests their chiasmal origin. Amongst these scotomata one encounters some which bear a strong resemblance to the nerve fibre bundle scotomata seen in optic nerve lesions (Fig. 10, overleaf). They mostly arise from the area of the blind spot though more central arcuate scotomata may be seen. I have not yet encountered a sector defect in the temporal field such as is seen in the optic nerve and retinal lesions. The occurrence of such scotomata is strongly suggestive of an individual and definitive blood supply to bundles of fibres just like that seen in the optic nerve, and one may presume that this fibre bundle system is continued for

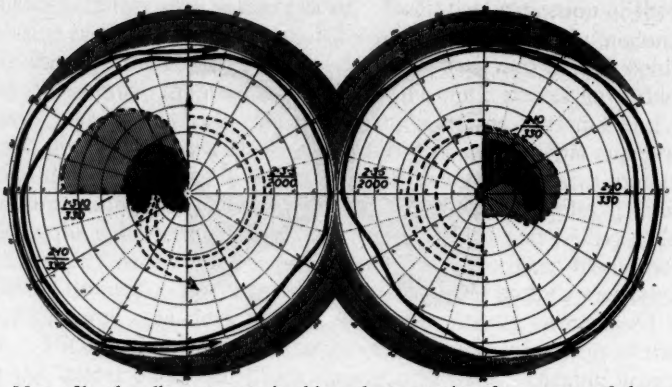

FIG. 10.—Nerve fibre bundle scotomata in chiasmal compression, from a case of chromophobe adenoma of the pituitary.

some way at least into the chiasma (Dubois-Poulsen, 1952). Most observers have noted, however, that the arteriolar and capillary pattern does not follow the fibre pattern in the chiasma. Most of such scotomata are in the central temporal field and yet the vascular pattern of the macular fibre area is as haphazard as anywhere in the chiasma. In my experience, most of such examples have been in patients with post-fixed chiasmata and long optic nerves, and this may well be a sign of such a variant, the field loss being in reality related to the anterior chiasmal angle in which the fibre bundle arrangement and vascular pattern is prolonged from the optic nerves.

Lastly, there is one aspect of chiasmal compression fields that is very difficult indeed to explain; the residual temporal island. In a small proportion of fields, usually those of a scotomatous character, the initial central scotoma breaks through to the upper and lower temporal periphery, isolating a small island of vision far out in the temporal field (Fig. 11). This

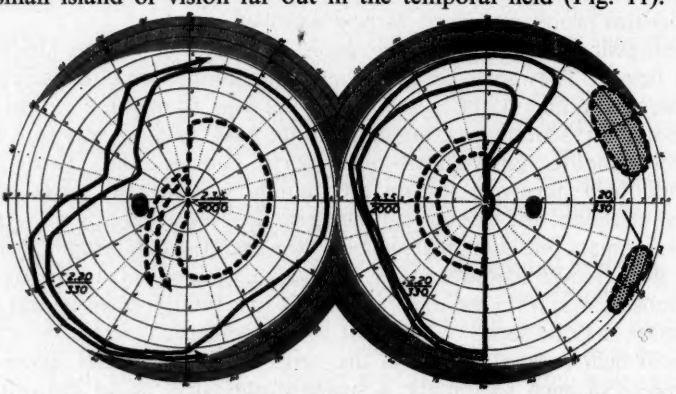

FIG. 11.—Temporal islands in chiasmal compression, from a case of chromophobe adenoma of the pituitary.

island tends to persist and may still be present long after the remainder of the temporal field has vanished and the nasal field become involved. These islands most often occur above or below the horizontal meridian and may represent the forward and backward loops of peripheral fibres in the chiasma. Traquair (1948) and Dubois-Poulsen (1952) both state that such islands are not seen when the lesion is prechiasmal or affecting the inner sides of the optic nerves, and yet examples following trauma have been reported. The only conceivable explanation is that the looping fibres are protected by their position from lesions affecting the antero-central area of the chiasma as far as the inferior field is concerned and the postero-central area as far as the upper field is concerned. Whether in these situations such fibres also enjoy a selective blood supply with the uncrossed fibres amongst which they lie is purely speculative. On the other hand such islands often cross the horizontal meridian and as such one would have to look for a situation in which the upper and lower peripheral fibres lie together. In the body of the chiasma itself, if we are to believe present concepts as to fibre anatomy, they lie as far apart as it is possible. The lesions in such cases, therefore, can only be in the exact centre of the chiasma.

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Brit. J. Ophthal. (1958) **42**, 126.

CORRESPONDENCE

INTRACAPSULAR CATARACT OPERATION

To the Editorial Committee of the BRITISH JOURNAL OF OPHTHALMOLOGY

SIRS—The recent article by Nirankari and Maudgal (1957) entitled "A Modification of the Smith Indian Technique of Intracapsular Cataract Operation" brings forth a very important point in modern cataract technique. This is a method of using extraction technique without a vitreous wedge, counter pressure being used only on the anterior segment of the eyeball and anterior to the vitreous.

No mention is made in this paper of pre-operative pressure on the eyeball. If pressure is exerted upon the eyeball for 5 minutes just before the operation, hypotony is usually obtained. With such hypotony the method described by these authors from India would not be applicable, as counter pressure above would not tilt the lens upwards from below to the degree desired.

As described by Harrington (1951), and stressed by the present writer (Hill, 1957), if the erisophake is used to lift the lower pole of the lens upwards, counter pressure can be placed higher up on the anterior segment—namely at the limbus or even anterior to the limbus. By this technique the pressure is farther forwards from the vitreous face, and counter pressure may be used to strip the tensed zonule from the capsule and the lens may be tumbled more easily and safely. The erisophake, if left in place on the capsule for 20 seconds, moulds the capsule into the cup—tensing the zonule fibres evenly along the circumference. The counter pressure is thereby more efficient in stripping away the attachment of the zonule to the anterior capsule and folding the cornea under the lens.

It is realized that the methods used in the large clinics of India involve a time element not considered in Western procedures, but the time consumed in placing the erisophake upon the lens for 20 seconds, which gives an added element of safety and further prevention of vitreous loss, is worth consideration. If necessary, this 5 minutes of pre-operative pressure may be exerted by a nurse or trained assistant.

Yours faithfully,
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BOOK REVIEWS

The Effect on Binocular Vision of Variations in the Relative Sizes and Levels of Illumination of the Ocular Images. By H. F. GILLOTT. 1957. Pp. 82, 34 figs, 50 refs. British Optical Association, London. (50*s.*)

When your reviewer discussed irradiation aniseikonia with the late Lord Charnwood, a few sporadic measurements showed that if it existed at all it must be a negligible effect.

This was, however, a somewhat glib conclusion. The only thing which can be questioned was the occurrence of irradiation: if this obtains, conditions for anomalous stereopsis exist. In an exhaustive study dealing with this and related topics, Gillott has shown that the effect indubitably exists, but that, under his conditions, it is small. His monograph covers the subject adequately from both an experimental and a theoretical point of view. His experiments were done on a large number of observers and are carefully analysed. A minor criticism refers to the amateurish lettering in the otherwise excellent figures, which detracts from the extrinsic neatness of the work, though not from its intrinsic value and interest.

Atlas of Eye Surgery. By R. T. PATON, H. M. KATZIN, and D. STILWELL. 1957. Pp. 248, 463 diagrams. McGraw-Hill, London. (£5 12*s.* 6*d.*)

The purpose of this atlas is to show the resident eye surgeon a basic technique which has as its aim safety in eight groups of eye operations: cataract, glaucoma, retinal detachment, corneal transplantation, enucleation, evisceration, squint, and pterygium. The authors have treated their matter admirably. The illustrations are excellent black-and-white line drawings which show clearly each step of an operation as seen by the operator—that is from above and behind the patient's head. These illustrations are clearly drawn and excellently designed and arranged to convey a pictorial sequence of the operative steps. The authors have indeed achieved their purpose.

It is hoped that subsequent editions may include operations on the lacrimal apparatus, lids, and orbit.

There is little to criticize. In the performance of iridocapsulotomy it is generally preferable to penetrate the iris and capsule with a stab from von Graefes cataract knife just before its withdrawal rather than to do so with the point of de Wecker's iris scissors as recommended in this book. The opening achieved by this operation is drawn as a triangle, but generally the triangular cut pupil becomes circular or almost so. The drawings of the diathermy operation and scleral resection for retinal detachment show diathermy applications unnecessarily extensive and wide of the retinal tear, and there is a misprint of 2–3 cm. for mm.

The condemnation of partially-exposed orbital implants is not firm enough.

Resection of the superior oblique is recommended as an alternative to pleating, but many surgeons would consider that the danger of resecting the long expanded tendon of this muscle is that the sutures may come asunder.

This atlas is of great value to the young surgeon, for upon the clear directions given by its admirable illustrations he could establish the basis of a sound and safe technique.

The publishers are to be congratulated on the excellence of its production.

Books Received

The following book has been received and will be the subject of a review in *Ophthalmic Literature*, **11**, No. 3, March, 1958.

Cortisone Therapy: Mainly Applied to the Rheumatic Diseases. By J. H. GLYN. 1957. Pp. 162, bibl. Heinemann, London. (21*s.*)

NOTES

In December, 1957, the Senate of the University of London conferred the title of Professor of Pathology in the University upon Dr. Norman Ashton in respect of his appointment as Director of the Department of Pathology at the Institute of Ophthalmology.

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OBITUARY

WILLIAM HEDLEY SUMMERSKILL

Bill Summerskill, as everybody knew him, died in Portsmouth after a long illness on Wednesday, November 13, 1957.

He was born in 1898 and, although the first world war, when he served in destroyers as probationary Surgeon Sub-Lieutenant, interrupted his medical training at Guy's, he qualified L.M.S.S.A. in 1920. He was in general practice in London from then until 1931, and during this time stood once as Liberal candidate for one of the London constituencies.

His career in ophthalmology started in 1931, in which year he was an undergraduate at Exeter College, Oxford. He held an appointment at the Oxford Eye Hospital, and took the D.O. (Oxon.) in 1933. He then practised in London, holding an appointment as clinical assistant to the Royal Eye Hospital, and qualifying M.B., B.S. During the second world war he served as P.M.O. in troopships, and was finally invalided home.

In 1943 he settled down to practise in Portsmouth, with an appointment as temporary ophthalmic surgeon to the Portsmouth and Southern Counties Eye and Ear Hospital. In 1944 he was appointed ophthalmic surgeon to the Royal West Sussex Hospital, Chichester. He was also visiting ophthalmic surgeon to the Queen Alexandra Ministry of Pensions Hospital at Cosham, and ophthalmic surgeon to St. Mary's Hospital, Portsmouth. The advent of the National Health Service saw his hospital appointments consolidated into an appointment as ophthalmic surgeon to the Portsmouth Group Hospitals which he held until his death.

His contributions to ophthalmology were many. He was an advocate of the transconjunctival approach to the orbit. He made an important modification of Mules's operation. His most notable contribution, however, was his work on intubation as a method of re-establishing lacrimal drainage, and his name, and his operation for this, are known all over the world. His contributions to ophthalmology were marked by the award of the Nathaniel Bishop Harman Prize in 1948, and he won the Treacher Collins Prize in 1957 for his essay on "Diseases of the Lacrimal Apparatus and their Treatment". He was president of the Southern Ophthalmological Society in 1955/56.

Summerskill was a man of great charm, and was always welcome where ophthalmologists gather. He made friends easily and had many, notably among his patients by whom he was much beloved. He had a robust and, at the same time, human approach to the sick, which contributed to the affection in which he was held. His chief interests in later life were sailing (he was chairman of the Royal Albert Yacht Club in 1952 and 1953) and the collection of old medical books. He came of a medical family. The son of a doctor, brother of another, and brother-in-law of a third, his son is a doctor, and he was uncle to doctors in the families of his two sisters. He will be greatly missed by all who knew him, and our deepest sympathy is extended to his widow, and to his son and daughter.